Minnifield
09/494297

- key terms
collagen-binding proteins

```
     FILE 'REGISTRY' ENTERED AT 10:13:02 ON 07 JUN 2001
                E COLLAGEN BINDING PROTEIN/CN
                E "COLLAGEN-BINDING PROTEIN"/CN
L1             1 S E4

     FILE 'CAPLUS' ENTERED AT 10:13:30 ON 07 JUN 2001
L2           139 S L1 OR COLLAGEN BIND? PROTEIN OR CPA#(S)COLLAGEN
L3             5 S L2 AND (STREPTOCOCC? OR PYOGENES)

L3   ANSWER 1 OF 5  CAPLUS  COPYRIGHT 2001 ACS
ACCESSION NUMBER:         2000:161169  CAPLUS
DOCUMENT NUMBER:          132:212703
TITLE:                    Multicomponent vaccines for prevention of
                          staphylococcal infections
INVENTOR(S):              Patti, Joseph M.; Foster, Timothy J.; Hook,
                          Magnus
PATENT ASSIGNEE(S):       Inhibitex, Inc., USA; The Texas A & M University
                          System; The Provost Fellows and Scholars of the
                          College of the Holy and Undivided Trinity of
                          Queen Elizabeth Near Dublin
SOURCE:                   PCT Int. Appl., 115 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.        KIND  DATE          APPLICATION NO.  DATE
     ---------------   ----  --------      ---------------  --------
     WO 2000012131      A1   20000309      WO 1999-US19727  19990831
         W:  AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR,
             CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU,
             ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,
             LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD,
             SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZA,
             ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
         RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE,
             DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ,
             CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG
     AU 9955889         A1   20000321      AU 1999-55889    19990831
PRIORITY APPLN. INFO.:                    US 1998-98439    P  19980831
                                          WO 1999-US19727  W  19990831
AB   Multicomponent vaccines are provided which aid in the prevention and
     treatment of staphylococcal infections and which include certain
     selected combinations of bacterial binding proteins or fragments
     thereof, or antibodies to those proteins or fragments.  By careful
     selection of the proteins, fragments, or antibodies, a vaccine is
     provided that imparts protection against a broad spectrum of
```

Staphylococcus bacterial strains and against proteins that are expressed at different stages of the logarithmic growth curve. In one embodiment of the invention, a compn. is provided that includes at least a **collagen-binding protein** or peptide (or an appropriate site directed mutated sequence thereof) such as CNA, or a protein or fragment with sufficiently high homol. thereto, in combination with a fibrinogen binding protein, preferably Clumping factor A ("ClfA") or Clumping factor B ("ClfB"), or a useful fragment thereof or a protein or fragment with sufficiently high homol. thereto. The vaccines and products of the present invention are advantageous in that they respond to the urgent need of the medical community for a substitute for small mol. antibiotics, which are rapidly losing effectiveness and provide effective combinations of the large no. of known bacterial surface adhesins which can impart effective protection against a broad spectrum of bacterial infections.

REFERENCE COUNT: 4
REFERENCE(S):
(1) Guss; US 5851794 A 1998 CAPLUS
(2) Hook; US 5440014 A 1995 CAPLUS
(3) Hook; US 5648240 A 1997 CAPLUS
(4) Wayner; US 5730978 A 1998 CAPLUS

L3 ANSWER 2 OF 5 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1999:159061 CAPLUS
DOCUMENT NUMBER: 131:14761
TITLE: Characterization of nra, a global negative regulator gene in group A **streptococci**
AUTHOR(S): Podbielski, Andreas; Woischnik, Markus; Leonard, Bettina A. B.; Schmidt, Karl-Hermann
CORPORATE SOURCE: Department of Medical Microbiology and Hygiene, University Hospital Ulm, Ulm, D-89081, Germany
SOURCE: Mol. Microbiol. (1999), 31(4), 1051-1064
CODEN: MOMIEE; ISSN: 0950-382X
PUBLISHER: Blackwell Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB During sequencing of an 11.5 kb genomic region of a serotype M49 group A **streptococcal** (GAS) strain, a series of genes were identified including nra (neg. regulator of GAS). Transcriptional anal. of the region revealed that nra was primarily monocistronically transcribed. Polycistronic expression was found for the three open reading frames (ORFs) downstream and for the four ORFs upstream of nra. The deduced Nra protein sequence exhibited 62% homol. to the GAS RofA pos. regulator. In contrast to RofA, Nra was found to be a neg. regulator of its own expression and that of the two adjacent operons by anal. of insertional inactivation mutants. By polymerase chain reaction and hybridization assays of 10 different GAS serotypes, the genomic presence of nra, rofA or

both was demonstrated. Nra-regulated genes include the fibronectin-binding protein F2 gene (prtF2) and a novel **collagen-binding protein** (**cpa**). The **Cpa** polypeptide was purified as a recombinant maltose-binding protein fusion and shown to bind type I **collagen** but not fibronectin. In accordance with nra acting as a neg. regulator of prtF2 and **cpa**, levels of attachment of the nra mutant strain to immobilized **collagen** and fibronectin was increased above wild-type levels. In addn., nra was also found to regulate neg. (four- to 16-fold) the global pos. regulator gene, mga. Using a strain carrying a chromosomally integrated duplication of the nra 3' end and an nra-luciferase reporter gene transcriptional fusion, nra expression was obsd. to reach its max. during late logarithmic growth phase, while no significant influence of atm. conditions could be distinguished clearly.

IT **225374-31-2**
RL: BPR (Biological process); PRP (Properties); BIOL (Biological study); PROC (Process)
(amino acid sequence; Nra-regulated genes include fibronectin-binding protein F2 gene prtF2 and **collagen-binding protein** gene **cpa** in group A **streptococci**)

REFERENCE COUNT: 53
REFERENCE(S):
(2) Brakhage, A; Biochimie 1990, V72, P725 CAPLUS
(3) Caparon, M; J Bacteriol 1992, V174, P5693 CAPLUS
(4) Caparon, M; Methods Enzymol 1991, V204, P556 CAPLUS
(5) Chen, C; Mol Gen Genet 1993, V241, P685 CAPLUS
(6) Chen, D; J Biol Chem 1994, V269, P32120 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1995:311506 CAPLUS
DOCUMENT NUMBER: 122:75071
TITLE: Isolation and characterization of a novel **collagen-binding protein** from **Streptococcus pyogenes** strain 6414
AUTHOR(S): Visai, Livia; Bozzini, Silvia; Raucci, Giuseppe; Toniolo, Antonio; Speziale, Pietro
CORPORATE SOURCE: Dep. Biochem., Univ. Pavia, Pavia, I-227100, Italy
SOURCE: J. Biol. Chem. (1995), 270(1), 347-53

CODEN: JBCHA3; ISSN: 0021-9258
DOCUMENT TYPE: Journal
LANGUAGE: English
AB In this report the authors have analyzed the binding of collagen to **Streptococcus pyogenes** strain 6414. This binding was rapid, specific, and involved a limited no. of receptor mols. (11,600 copies per cell). When the proteins in a **streptococcal** lysate were blotted onto a nitrocellulose filter and probed with 125I-labeled collagen, a prominent **collagen-binding protein** of 57 kDa was identified as well as minor 130-150-kDa components. The major 57-kDa protein was isolated by affinity chromatog. on collagen-Sepharose followed by gel filtration chromatog. The 57-kDa protein purified from S. **pyogenes** was used to raise a monospecific antibody which also reacted with a **collagen-binding protein** of similar mol. size isolated from **Streptococcus** zooepidemicus. The two **collagen-binding proteins** from **streptococci** have a similar amino acid compn. and isoelec. points. Isolated **collagen-binding protein** was specifically recognized by 125I-collagen in a solid-phase binding assay and displayed an affinity for the ligand quite similar to that exhibited by intact bacteria (Kd = 3.1 vs. 3.5.times.10-9 M, resp.). Surface-labeled bacteria attached to microtiter wells coated with different collagen types and the 57-kDa protein blocked the adhesion to collagen substrate. The authors propose that the 57-kDa protein is an adhesin involved in the attachment of **streptococci** to host tissues.

L3 ANSWER 4 OF 5 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1994:51458 CAPLUS
DOCUMENT NUMBER: 120:51458
TITLE: Collagen mediates adhesion of **Streptococcus** mutans to human dentin
AUTHOR(S): Switalski, Lech M.; Butcher, Wade G.; Caufield, Page C.; Lantz, Marilyn S.
CORPORATE SOURCE: Sch. Dent. Med., Univ. Pittsburgh, Pittsburgh, PA, 15261, USA
SOURCE: Infect. Immun. (1993), 61(10), 4119-25
CODEN: INFIBR; ISSN: 0019-9567
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Some strains of **Streptococcus** mutans were found to recognize and bind collagen type I. Binding of 125I-labeled collagen type I was specific in heat collagen types I and II, but not unrelated proteins, were able to inhibit binding to the labeled ligand to bacteria. Collagen binding to S. mutans was partially reversible and involved a limited no. of bacterial binding sites per

cell. S. mutans UA 140 cells bound collagen type I with high affinity. The no. of binding sites per cell was 4 .times. 104. Collagen-binding strains of S. mutans adhered to collagen-coated surfaces as well as to pulverized root tissue. S. mutans strains that did not bind the sol. ligand were unable to adhere to these substrata. Adherence to collagen-coated surfaces could be inhibited with collagen or clostridial collagenase-derived collagen peptides. S. mutants UA 140 bound significantly less 125I-collagen type I following treatment with peptidoglycan-degrading enzymes. These enzymes released a **collagen-binding protein** (collagen receptor) with a relative mol. size of 16 kDa. Apparently, collagen mediates adhesion of S. mutans to dentin. This interaction may target collagen-binding strains of S. mutans to dentin in the oral cavity and may play a role in the pathogenesis of root surface caries.

L3 ANSWER 5 OF 5 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1993:666019 CAPLUS
DOCUMENT NUMBER: 119:266019
TITLE: Bacterial proteins binding to the mammalian extracellular matrix
AUTHOR(S): Westerlund, B.; Korhonen, T. K.
CORPORATE SOURCE: Dep. Gen. Microbiol., Univ. Helsinki, Helsinki, SF-00014, Finland
SOURCE: Mol. Microbiol. (1993), 9(4), 687-94
CODEN: MOMIEE; ISSN: 0950-382X
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review with 73 refs. Pathogenic bacteria frequently express surface proteins with affinity for components of the mammalian extracellular matrix, i.e. collagens, laminin, fibronectin or proteoglycans. This review summarizes the authors' current knowledge on the mechanisms of bacterial adherence to extracellular matrixes and on the biol. significance of these interactions. The best-characterized bacterial proteins active in these interactions are the mycobacterial fibronectin-binding proteins, the fibronectin- and the **collagen-binding proteins** of staphylococci and **streptococci**, specific enterobacterial fimbrial types, as well as the polymeric surface proteins YadA of yersinias and the A-protein of Aeromonas. Some of these bacterial proteins are highly specific for an extracellular matrix protein, some are multifunctional and express binding activities towards a no. of target proteins. The interactions can be based on a protein-protein or on a protein-carbohydrate interaction, or on a bridging mechanism mediated by a bivalent sol. target protein. Many of the interactions have also been demonstrated on tissue sections or in vivo, and adherence to the extracellular matrix has been shown to promote bacterial colonization of damaged tissues.

(FILE 'CAPLUS' ENTERED AT 10:13:30 ON 07 JUN 2001)
L4 5 S L2 AND (STREPTOCOCC? OR PYOGENES OR GAS(S)STREPTOCOCC?)
L5 0 S L4 NOT L3

(FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, CONFSCI, SCISEARCH, JICST-EPLUS, JAPIO' ENTERED AT 10:15:34 ON 07 JUN 2001)
L6 19 S L4
L7 7 DUP REM L6 (12 DUPLICATES REMOVED)

L7 ANSWER 1 OF 7 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD
ACCESSION NUMBER: 2000-237781 [20] WPIDS
DOC. NO. CPI: C2000-072441
TITLE: Composition used for generating immune response or for inhibiting microbial colonization in an animal comprises antibodies that bind **collagen binding protein**, fibrinogen binding protein and, optionally, fibronectin binding protein.
DERWENT CLASS: B04 D16
INVENTOR(S): FOSTER, T J; HOOK, M; PATTI, J M
PATENT ASSIGNEE(S): (INHI-N) INHIBITEX INC; (QUEE-N) QUEEN ELIZABETH COLLEGE DUBLIN; (TEXA) UNIV TEXAS A & M SYSTEM
COUNTRY COUNT: 87
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2000012131	A1	20000309	(200020)*	EN	115

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ UG ZW
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG UZ VN YU ZA ZW

AU 9955889	A	20000321 (200031)	

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000012131	A1	WO 1999-US19727	19990831
AU 9955889	A	AU 1999-55889	19990831

FILING DETAILS:

PATENT NO	KIND	PATENT NO

AU 9955889 A Based on WO 200012131

PRIORITY APPLN. INFO: US 1998-98439 19980831
AN 2000-237781 [20] WPIDS
AB WO 200012131 A UPAB: 20000426
NOVELTY - A composition (I) comprising an antibody that binds to a collagen binding (CB) domain of a CB protein, inhibiting binding to collagen, an antibody that binds to a fibrinogen binding (FB) domain of a FB protein, inhibiting binding to fibrinogen, and optionally an antibody that binds to a fibronectin binding (FnB) domain of a FnB protein, inhibiting binding to fibronectin, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a vaccine (II) comprising the CB domain of the CB protein, the FB domain of the FB protein and, optionally, the FnB domain of the FnB protein;

(2) a vaccine (III) comprising the fibronectin ligand peptide that would be bound by an FnB protein, the collagen ligand peptide that would be bound by a CB protein and the fibrinogen ligand peptide that would be bound by an FB protein;

(3) a method of generating an immune response comprising administering (II) to an animal;

(4) a vaccine (IV) comprising nucleic acid encoding a CB protein, nucleic acid encoding an FB protein and, optionally, a nucleic acid encoding an FnB protein; and

(5) a vaccine (V) comprising the FnB proteins.

ACTIVITY - Immunostimulatory; antibacterial.

Sixty female Swiss Webster mice received a total of 50 micro g of either ovalbumin, M55 (collagen-binding MSCRAMM (microbial surface components recognizing adhesive matrix molecules)) or a combination of M55 and ClfA (fibrinogen-binding MSCRAMM) proteins via a subcutaneous injection. The primary injection was prepared by emulsifying the antigens in Freund's Complete Adjuvant. The mice received a second injection of 25 micro g total protein in Freund's Incomplete Adjuvant 14 days after the primary injection. A final injection of 25 micro g total protein in PBS (phosphate buffered solution) was given 28 days after the primary injection. Post bleed samples from all mice were obtained two weeks after the final injection to determine antibody titers against the different MSCRAMM proteins. The mice were then challenged (42 days after primary injection) via a single intravenous injection with 1.2 X 108 colony forming units (CFU) of Staphylococcus aureus 601. At day 5 post-challenge, the mice were sacrificed and their kidneys harvested. The kidneys were then homogenized and plated on blood agar plates. The plates were incubated at 37 deg. C overnight and the bacterial load in the kidneys was determined by colony counts. The results of the experiment showed a two log difference in bacterial load between the ovalbumin group (7.03 plus or minus 0.93

log CFU/g) and the M55/ClfA group (4.83 plus or minus 3.04 log CFU/g, p = 0.006). A difference in bacterial load was also observed in the M55 group (5.86 plus or minus 3.42 log CFU/g, p = 0.003) when compared to the ovalbumin group.

MECHANISM OF ACTION - Vaccine.

USE - The composition, vaccines and method are useful for generating an immune response in an animal and for inhibiting microbial colonization, especially Staphylococcus aureus, in an animal. The proteins are used in active vaccines and the antibodies in passive vaccines. The combinations can also be used to select donor blood pools for the preparation of purified blood products for passive immunization.

Dwg.0/5

L7 ANSWER 2 OF 7 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 1999195808 MEDLINE
DOCUMENT NUMBER: 99195808 PubMed ID: 10096074
TITLE: Characterization of nra, a global negative regulator gene in group A **streptococci**.
AUTHOR: Podbielski A; Woischnik M; Leonard B A; Schmidt K H
CORPORATE SOURCE: Department of Medical Microbiology and Hygiene, University Hospital Ulm, Germany.. andreas.podbielski@medizin.uni-ulm.de
SOURCE: MOLECULAR MICROBIOLOGY, (1999 Feb) 31 (4) 1051-64. Journal code: MOM; 8712028. ISSN: 0950-382X.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199907
ENTRY DATE: Entered STN: 19990727
Last Updated on STN: 19990727
Entered Medline: 19990715

AB During sequencing of an 11.5 kb genomic region of a serotype M49 group A **streptococcal** (**GAS**) strain, a series of genes were identified including nra(negative regulator of **GAS**). Transcriptional analysis of the region revealed that nra was primarily monocistronically transcribed. Polycistronic expression was found for the three open reading frames (ORFs) downstream and for the four ORFs upstream of nra. The deduced Nra protein sequence exhibited 62% homology to the **GAS** RofA positive regulator. In contrast to RofA, Nra was found to be a negative regulator of its own expression and that of the two adjacent operons by analysis of insertional inactivation mutants. By polymerase chain reaction and hybridization assays of 10 different **GAS** serotypes, the genomic presence of nra, rofA or both was demonstrated. Nra-regulated genes include the fibronectin-binding protein F2 gene (prtF2) and a novel **collagen**-

binding protein (**cpa**). The **Cpa** polypeptide was purified as a recombinant maltose-binding protein fusion and shown to bind type I **collagen** but not fibronectin. In accordance with nra acting as a negative regulator of prtF2 and **cpa**, levels of attachment of the nra mutant strain to immobilized **collagen** and fibronectin was increased above wild-type levels. In addition, nra was also found to regulate negatively (four- to 16-fold) the global positive regulator gene, mga. Using a strain carrying a chromosomally integrated duplication of the nra 3' end and an nra-luciferase reporter gene transcriptional fusion, nra expression was observed to reach its maximum during late logarithmic growth phase, while no significant influence of atmospheric conditions could be distinguished clearly.

L7 ANSWER 3 OF 7 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 1999:10644 SCISEARCH
THE GENUINE ARTICLE: 148FC
TITLE: Surface protein adhesins of Staphylococcus aureus
AUTHOR: Foster T J (Reprint); Hook M
CORPORATE SOURCE: TRINITY COLL DUBLIN, DEPT MICROBIOL, MOYNE INST PREVENT MED, DUBLIN 2, IRELAND (Reprint); TEXAS A&M UNIV, CTR EXTRACELLULAR MATRIX BIOL, HOUSTON, TX 77030; TEXAS A&M UNIV, DEPT BIOCHEM & BIOPHYS, INST BIOSCI & TECHNOL, HOUSTON, TX 77030
COUNTRY OF AUTHOR: IRELAND; USA
SOURCE: TRENDS IN MICROBIOLOGY, (DEC 1998) Vol. 6, No. 12, pp. 484-488.
Publisher: ELSEVIER SCI LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD OX5 1GB, OXON, ENGLAND.
ISSN: 0966-842X.
DOCUMENT TYPE: General Review; Journal
LANGUAGE: English
REFERENCE COUNT: 48
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Staphylococcus aureus can colonize the host to initiate infection by adhering to components of the extracellular matrix., Adherence is mediated by surface protein adhesins (MSCRAMMs). Ligand binding by these fibronectin-, fibrinogen- and **collagen-binding proteins** occurs by distinct mechanisms that are being investigated at the molecular level.

L7 ANSWER 4 OF 7 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 95113849 MEDLINE
DOCUMENT NUMBER: 95113849 PubMed ID: 7814395
TITLE: Isolation and characterization of a novel **collagen-binding protein** from **Streptococcus pyogenes** strain 6414.

AUTHOR: Visai L; Bozzini S; Raucci G; Toniolo A; Speziale P
CORPORATE SOURCE: Department of Biochemistry, University of Pavia, Italy.
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1995 Jan 6) 270 (1) 347-53.
Journal code: HIV; 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199502
ENTRY DATE: Entered STN: 19950217
Last Updated on STN: 19950217
Entered Medline: 19950203

AB In this report we have analyzed the binding of collagen to **Streptococcus pyogenes** strain 6414. This binding was rapid, specific, and involved a limited number of receptor molecules (11,600 copies per cell). When the proteins in a **streptococcal** lysate were blotted onto a nitrocellulose filter and probed with 125I-labeled collagen, a prominent **collagen-binding protein** of 57 kDa was identified as well as minor 130-150-kDa components. The major 57-kDa protein was isolated by affinity chromatography on collagen-Sepharose followed by gel filtration chromatography. The 57-kDa protein purified from S. **pyogenes** was used to raise a monospecific antibody which also reacted with a **collagen -binding protein** of similar molecular size isolated from **Streptococcus** zooepidemicus. The two **collagen-binding proteins** from **streptococci** have a similar amino acid composition and isoelectric points. Isolated **collagen-binding protein** was specifically recognized by 125I-collagen in a solid-phase binding assay and displayed an affinity for the ligand quite similar to that exhibited by intact bacteria (Kd = 3.1 versus 3.5 x 10(-9) M, respectively). Surface-labeled bacteria attached to microtiter wells coated with different collagen types and the 57-kDa protein blocked the adhesion to collagen substrate. We propose that the 57-kDa protein is an adhesin involved in the attachment of **streptococci** to host tissues.

L7 ANSWER 5 OF 7 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 94011294 MEDLINE
DOCUMENT NUMBER: 94011294 PubMed ID: 8406800
TITLE: Collagen mediates adhesion of **Streptococcus** mutans to human dentin.
AUTHOR: Switalski L M; Butcher W G; Caufield P C; Lantz M S
CORPORATE SOURCE: Department of Periodontics, School of Dental Medicine, University of Pittsburgh, Pennsylvania

15261.
CONTRACT NUMBER: DE 07256 (NIDCR)
DE 09082 (NIDCR)
DE 10397 (NIDCR)
SOURCE: INFECTION AND IMMUNITY, (1993 Oct) 61 (10) 4119-25.
Journal code: GO7; 0246127. ISSN: 0019-9567.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199311
ENTRY DATE: Entered STN: 19940117
Last Updated on STN: 20000303
Entered Medline: 19931116

AB Some strains of **Streptococcus** mutans were found to recognize and bind collagen type I. Binding of 125I-labeled collagen type I was specific in that collagen types I and II, but not unrelated proteins, were able to inhibit binding of the labeled ligand to bacteria. Collagen binding to S. mutans was partially reversible and involved a limited number of bacterial binding sites per cell. S. mutans UA 140 cells bound collagen type I with high affinity (Kd = 8 x 10(-8) M). The number of binding sites per cell was 4 x 10(4). Collagen-binding strains of S. mutans were found to adhere to collagen-coated surfaces as well as to pulverized root tissue. S. mutans strains that did not bind the soluble ligand were unable to adhere to these substrata. Adherence to collagen-coated surfaces could be inhibited with collagen or clostridial collagenase-derived collagen peptides. Adherence of S. mutans to dentin was enhanced by collagen types I and II but inhibited by collagen peptides. S. mutans UA 140 bound significantly less 125I-collagen type I following treatment with peptidoglycan-degrading enzymes. These enzymes released a **collagen-binding protein** (collagen receptor) with a relative molecular size of 16 kDa. The results of this study suggest that collagen mediates adhesion of S. mutans to dentin. This interaction may target collagen-binding strains of S. mutans to dentin in the oral cavity and may play a role in the pathogenesis of root surface caries.

L7 ANSWER 6 OF 7 MEDLINE DUPLICATE 4

ACCESSION NUMBER: 94049110 MEDLINE
DOCUMENT NUMBER: 94049110 PubMed ID: 7901732
TITLE: Bacterial proteins binding to the mammalian extracellular matrix.
AUTHOR: Westerlund B; Korhonen T K
CORPORATE SOURCE: Department of General Microbiology, University of Helsinki, Finland.
SOURCE: MOLECULAR MICROBIOLOGY, (1993 Aug) 9 (4) 687-94.

Ref: 73
Journal code: MOM; 8712028. ISSN: 0950-382X.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199312
ENTRY DATE: Entered STN: 19940117
Last Updated on STN: 19950206
Entered Medline: 19931221

AB Pathogenic bacteria frequently express surface proteins with affinity for components of the mammalian extracellular matrix, i.e. collagens, laminin, fibronectin or proteoglycans. This review summarizes our current knowledge on the mechanisms of bacterial adherence to extracellular matrices and on the biological significance of these interactions. The best-characterized bacterial proteins active in these interactions are the mycobacterial fibronectin-binding proteins, the fibronectin- and the **collagen-binding proteins** of staphylococci and **streptococci**, specific enterobacterial fimbrial types, as well as the polymeric surface proteins YadA of yersinias and the A-protein of Aeromonas. Some of these bacterial proteins are highly specific for an extracellular matrix protein, some are multifunctional and express binding activities towards a number of target proteins. The interactions can be based on a protein-protein or on a protein-carbohydrate interaction, or on a bridging mechanism mediated by a bivalent soluble target protein. Many of the interactions have also been demonstrated on tissue sections or in vivo, and adherence to the extracellular matrix has been shown to promote bacterial colonization of damaged tissues.

L7 ANSWER 7 OF 7 SCISEARCH COPYRIGHT 2001 ISI (R)

ACCESSION NUMBER: 92:136894 SCISEARCH
THE GENUINE ARTICLE: HF642
TITLE: MOLECULAR CHARACTERIZATION AND EXPRESSION OF A GENE ENCODING A STAPHYLOCOCCUS-AUREUS COLLAGEN ADHESIN
AUTHOR: PATTI J M (Reprint); JONSSON H; GUSS B; SWITALSKI L M; WIBERG K; LINDBERG M; HOOK M
CORPORATE SOURCE: UNIV ALABAMA, DEPT BIOCHEM, BIRMINGHAM, AL, 35294 (Reprint); UNIV ALABAMA, DEPT MICROBIOL, BIRMINGHAM, AL, 35294; SWEDISH UNIV AGR SCI, DEPT MICROBIOL, S-75007 UPPSALA, SWEDEN
COUNTRY OF AUTHOR: USA; SWEDEN
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (05 MAR 1992) Vol. 267, No. 7, pp. 4766-4772.
ISSN: 0021-9258.

DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 39
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Some strains of Staphylococcus aureus bind collagen with a high degree of specificity and affinity. This interaction can represent a mechanism of substrate adhesion and may be an important step in the pathogenesis of osteomyelitis and infectious arthritis. We now report on the cloning, sequencing, and expression of a gene named cna, encoding a S. aureus collagen adhesin. The cna gene was isolated from a lambda-GT11 S. aureus genomic library and encodes an 1185 amino acid polypeptide. The deduced amino acid sequence reveals several structural characteristics similar to previously described Gram-positive bacterial cell surface proteins. Antibodies raised against the native collagen adhesin from S. aureus recognize the recombinant collagen adhesin. Collagen binding activity can be detected in a lysate obtained from Escherichia coli cells, which harbor the cloned cna gene on an expression plasmid. **Collagen-binding proteins** can be detected in the lysate when analyzed by a Western blot type assay in which the membrane-transferred proteins are probed with radioactively labeled collagen. Finally, the bacterial lysate containing the recombinant adhesin can effectively inhibit the binding of soluble collagen to cells of S. aureus.

streptococcal infection

FILE 'REGISTRY' ENTERED AT 10:18:09 ON 07 JUN 2001
E COLLAGEN/CN
L8 334 SEA ABB=ON PLU=ON COLLAGEN ?/CN

FILE 'CAPLUS' ENTERED AT 10:18:35 ON 07 JUN 2001
L9 70333 SEA ABB=ON PLU=ON L8 OR COLLAGEN
L10 22 SEA ABB=ON PLU=ON L9 AND ((STREPTOCOCC? OR PYOGENES OR GAS(S)STREPTOCOCC?)(S)INFECTION)
L11 11 SEA ABB=ON PLU=ON L10 AND (TREAT? OR PROPHYL? OR THERAP? OR PREVENT?)
L12 10 SEA ABB=ON PLU=ON L11 NOT L3

L12 ANSWER 1 OF 10 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 2000:441821 CAPLUS
DOCUMENT NUMBER: 133:69838
TITLE: Novel fibronectin-binding protein SFS and corresponding gene of Streptococcus equi and use in vaccine preparation
INVENTOR(S): Guss, Bengt; Lindmark, Hans; Jacobsson, Karin; Frykberg, Lars
PATENT ASSIGNEE(S): Swed.
SOURCE: PCT Int. Appl., 34 pp.

CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000037496	A1	20000629	WO 1999-SE2448	19991221

W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG

PRIORITY APPLN. INFO.: SE 1998-4491 A 19981222

AB The present invention is concerned with a novel fibronectin-binding protein termed SFS of Streptococcus equi, and with the corresponding gene designated sfs. The SFS protein with a calcd. mol. mass of 40 kDA has a signal peptide with a possible cleavage site between amino acids 29 and 30, resulting in mature protein of 36 kDa. The protein SFS displays sequence similarity to both **collagen** and a potential cell-wall protein of S. pyogenes. The invention is related to host cells and vectors contg. sfs gene fragment and to methods to produce SFS protein based on recombinant DNA technol. The invention is also related to use of said protein in the prepn. of a vaccine, to a vaccine contg. said protein, to antibodies specific for said protein and to polyvalent antisera contg. such antibodies.

REFERENCE COUNT: 3
REFERENCE(S):
(1) Hans, L; Infection and Immunity 1996, V64(10), P3993
(2) Hans, L; Infection and Immunity 1999, V67(5), P2383
(3) The Texas A & M University System; WO 9831389 A2 1998 CAPLUS

L12 ANSWER 2 OF 10 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1998:618371 CAPLUS
DOCUMENT NUMBER: 129:255004
TITLE: **Prophylactic** and **therapeutic** methods for ocular degenerative diseases and inflammations, and histidine compositions therefor
INVENTOR(S): Thomas, Peter G.

PATENT ASSIGNEE(S): Cytos Pharmaceuticals LLC, USA
SOURCE: U.S., 10 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5811446	A	19980922	US 1997-839805	19970418
WO 9847366	A1	19981029	WO 1998-US7319	19980417
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9873583	A1	19981113	AU 1998-73583	19980417
PRIORITY APPLN. INFO.:			US 1997-839805	19970418
			WO 1998-US7319	19980417

AB Methods are provided for protecting the eye from degenerative eye conditions by administering **prophylactic** histidine compns. Also provided are for **treating** ocular inflammation resulting from various causative agents, by administering **therapeutic** histidine compns. Further provided are histidine compns. for carrying out the methods.

IT **9001-12-1**, Collagenase
RL: BSU (Biological study, unclassified); BIOL (Biological study) (inhibitors; histidine compns. and methods for ocular degenerative diseases and inflammations)

L12 ANSWER 3 OF 10 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1996:653321 CAPLUS
DOCUMENT NUMBER: 125:284881
TITLE: Bacteria presenting binding sites for viruses and capable of colonizing mucosal membranes for protection against viral infection
INVENTOR(S): Lee, Peter Poon-hang
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 36 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1

PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9627292	A1	19960912	WO 1996-US3151	19960308
W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI				
RW: KE, LS, MW, SD, SZ, UG, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN				
US 5733540	A	19980331	US 1995-401070	19950308
CA 2214723	AA	19960912	CA 1996-2214723	19960308
AU 9653044	A1	19960923	AU 1996-53044	19960308
AU 696449	B2	19980910		
BR 9607146	A	19971125	BR 1996-7146	19960308
EP 871363	A1	19981021	EP 1996-909613	19960308
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
JP 11501632	T2	19990209	JP 1996-527052	19960308

PRIORITY APPLN. INFO.: US 1995-401070 19950308
WO 1996-US3151 19960308

AB A method of using genetically modified, non-pathogenic bacteria on the mucosal surfaces of a host to inhibit infection by specific viruses at mucosal surfaces is described. Specifically, non-pathogenic bacteria are modified to acquire the capacity to bind and functionally inactivate specific viruses. The binding may be achieved through the natural binding site for the virus or with an antibody to the virus. Further manipulations are devised to ensure the persistent colonization of said bacteria on the desired mucosal surface of a host. The capacity to bind a pathogen may be obtained through the presentation on the bacterial surface of a mol., either a polypeptide or carbohydrate moiety, which binds specifically to a mol. on the target virus. The method is demonstrated in vitro by showing that Staphylococcus aureus bearing the human rhinovirus-binding domain of ICAM-1 **prevented** the binding of the virus to human lung fibroblasts.

IT **9001-12-1**, Collagenase
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(modification of mucosal surface with, for improved bacterial adhesion; bacteria presenting binding sites for viruses and capable of colonizing mucosal membranes for protection against viral infection)

L12 ANSWER 4 OF 10 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1996:614684 CAPLUS
DOCUMENT NUMBER: 125:298695

TITLE: I. A subpopulation of human urothelial cells is stimulated to proliferate by **treatment** in vitro with lipoteichoic acid, a cell wall component of Streptococcus faecalis

AUTHOR(S): Elgavish, Ada; Lloyd, Keith; Reed, Rebecca
CORPORATE SOURCE: Medical School, University of Alabama at Birmingham, Birmingham, AL, 35294, USA

SOURCE: J. Cell. Physiol. (1996), 169(1), 42-51
CODEN: JCLLAX; ISSN: 0021-9541
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Urinary tract infection with gram-pos. bacteria is common. Avenues for ingress of bacteria into the bladder include luminal and suburothelial infection. Terminally differentiated superficial urothelial cells lining the lumen of the bladder are often shed in response to infection. In contrast, infection-induced altered function of progenitors of urothelial cells residing in the basal layer of the urothelium is likely to have long lasting effects on the structure and function of the urothelium. The main objective of the present studies was to investigate in vitro the possibility that exposure to lipoteichoic acid, a cell wall component of the gram-pos. Streptococcus faecalis (LT-2), stimulates basal urothelial cells to proliferate. To simulate conditions that restrict proliferation and inhibit terminal differentiation of urothelial cells in the basal layer, secondary cultures of urothelial cells (UT) were grown on **collagen** or fibronectin-coated substrate in medium contg. low levels of Ca2+ (0.2 mM) and growth factors (0.005% bovine pituitary ext. [BPE]). Under these conditions, UT cultures displayed a highly reproducible colony size distribution, possibly due to the fact that colonies were progeny of basal cells with various proliferative potentials, retained in vitro. In cultures grown under growth-restricting conditions, the majority of progenitors appeared to be quiescent, just like stem cells in the basal layer of the urothelium. Thus, the population of large colonies (more than six cells/colony), was small when a steady state of growth was achieved, 3-7 days after seeding. Growth factors (0.005-0.5% BPE) caused a dose-dependent increase in this population of large colonies. Moreover, **treatment** of UT grown under growth-restricting conditions (0.005% BPE) with LT-2 increased steady-state levels of the population of large colonies to levels obtained in cultures growing under optimal conditions with respect to growth factors. These results indicated that the subpopulation of progenitors, quiescent under normal conditions, could be stimulated to proliferate. Two lines of evidence were consistent with the possibility that **treatment** with LT-2 stimulated proliferation of the subpopulation of progenitors and that large colonies were the progeny of this subpopulation of single cells: (1) **treatment** with LT-2 increased the percentage of

single cells that incorporated bromodeoxyuridine (i.e., proliferated) in a time-dependent manner; (2) an increase in the percentage of large colonies was found following LT-2-triggered proliferation of single cells. The authors propose that, under normal conditions, cells produced in response to LT-2-triggered proliferation of stem cells are removed from the system due to an increased rate of differentiation followed by apoptosis. Recurrent infection and inflammation may not allow these processes to proceed effectively, resulting in chronic injury to the bladder. Moreover, under conditions in which stem cells accumulate mutations that incapacitate their progeny to undergo apoptosis, LT-triggered proliferation could be a contributing factor to tumorigenesis.

L12 ANSWER 5 OF 10 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1994:564030 CAPLUS
DOCUMENT NUMBER: 121:164030
TITLE: Methods and compositions for the direct concentrated delivery of passive immunity
INVENTOR(S): Gristina, Anthony George
PATENT ASSIGNEE(S): USA
SOURCE: PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9415640	A1	19940721	WO 1994-US410	19940111
W: CA, JP				
RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
CA 2153661	AA	19940721	CA 1994-2153661	19940111
EP 680337	A1	19951108	EP 1994-909448	19940111
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE				
JP 08508240	T2	19960903	JP 1994-516308	19940111

PRIORITY APPLN. INFO.: US 1993-3305 A 19930112
WO 1994-US410 W 19940111

AB Compns. contg. a high concn. of the full repertoire of Igs, including IgA, IgM and IgG, are used to combat infections from microorganisms and viruses at a wound, surgical, or burn site, or normal tissue at times of risk of infection. The compns. can contain elevated antibody titers for several specific pathogens including Staphylococcus aureus, coagulase-neg. Staphylococci, Enterococci, S. epidermis, Pseudomonas aeruginosa, Escherichia coli, and Enterobacter sp., etc. The compns. are applied directly to a

wound or burn site as an ointment, cream, fluid, spray, or the like, prior to viral or bacterial attachment or biofilm formation such that adhesion of the pathogens is inhibited and the pathogens closest to the wound or burn site will be pre-opsonized for phagocytic killing prior to toxic release. The Igs in the compn. can be immobilized on a biocompatible material such as **collagen**, fibrin, hyaluronan, biodegradable polymers, and fragments thereof, which will be placed in-situ at the wound, surgical or burn site. In addn., the Igs in the compn. may be coated on the body contacting surface of an implantable device such as a catheter, contact lens or total joint.

L12 ANSWER 6 OF 10 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1993:446353 CAPLUS
DOCUMENT NUMBER: 119:46353
TITLE: Transforming growth factor-.beta. and the fibrotic response
AUTHOR(S): Flanders, K. C.
CORPORATE SOURCE: Natl. Cancer Inst., Natl. Inst. Health, Bethesda, MD, 20882, USA
SOURCE: Mol. Cell Biol. Liver Fibrogenesis, Proc. Int. Falk Symp. (1992), 241-53. Editor(s): Gressner, A. M.; Ramadori, G. Kluwer: Dordrecht, Neth. CODEN: 58ZZAF
DOCUMENT TYPE: Conference; General Review
LANGUAGE: English
AB A review with 49 refs. Transforming growth factor-.beta. (TGF-.beta.) was first identified as a protein with the ability to induce colony formation of some cells in soft agar. It is now known to be a multifunctional factor with a widespread distribution in adult tissues, as well as in embryonic development. Three distinct isoforms of TGF-.beta. exhibiting 70-80% sequence homol. are expressed in mammals. All three isoforms seem to be secreted from cells in a latent complex which is unable to bind to cell surface receptors and elicit biol. responses. It is postulated that the conversion of latent TGF-.beta. to its active form is a crit. step in regulating the many biol. actions of TGF-.beta.. Several of the reported biol. actions of TGF-.beta. might contribute to fibrosis in the liver. In vitro, TGF-.beta. **treatment** induces prodn. of a variety of extracellular matrix proteins in many cell types, and it has been shown to increase secretion of **collagen** and proteoglycans from activated lipocytes. It also is chemotactic for monocytes and induces macrophages to secrete addnl. TGF-.beta., as well as other cytokines, which may contribute to fibrosis in the inflammatory stage of disease. Activated lipocytes and Kupffer cells also have been shown to secrete TGF-.beta., and auto-induction of TGF-.beta. in activated lipocytes may be a continuing source of TGF-.beta. in chronic fibrosis. There is a transient increase in

TGF-.beta. mRNA levels in regenerating liver following partial hepatectomy which is followed by a transient increase in extracellular matrix proteins. However, an increase in TGF-.beta. protein has not been detected. Hepatocytes from regenerating liver more readily activate latent TGF-.beta. and arrest their proliferation than hepatocytes from normal liver, suggesting that TGF-.beta. may contribute to halting the proliferative phase of regeneration. Parallel increases of TGF-.beta.1 mRNA and **collagen** mRNA are seen using a no. of models of hepatic fibrosis, including CCl4 administration, schistosomiasis **infection**, and i.p. injection of **streptococcal** cell walls. In situ hybridization shows expression of TGF-.beta. in inflammatory cells and perisinusoidal cells, and increased expression of immunoreactive TGF-.beta. is obsd. in invading mononuclear cells, Kupffer cells, as well as hepatocytes. Immunoreactive TGF-.beta. in parenchymal cells probably represents increased amts. of TGF-.beta. taken up by these cells. Livers of patients with cirrhosis also show higher levels of immunoreactive TGF-.beta., as well as increased expression of TGF-.beta.1 and **collagen** mRNAs, compared to normal livers. In some patients interferon **treatment** decreased fibrosis, as well as levels of TGF-.beta. mRNA. These data suggest that increased levels of TGF-.beta., possibly produced by inflammatory cells and activated lipocytes, contribute to hepatic fibrosis.

L12 ANSWER 7 OF 10 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1991:639827 CAPLUS
DOCUMENT NUMBER: 115:239827
TITLE: Device and method for extended delivery of pharmacologically active agents to the middle ear
INVENTOR(S): Muchow, David C.; Sirvio, Larry M.
PATENT ASSIGNEE(S): Minnesota Mining and Mfg. Co., USA
SOURCE: Eur. Pat. Appl., 16 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 447719	A1	19910925	EP 1990-313800	19901218
EP 447719	B1	19931103		

R: CH, DE, FR, GB, LI, SE

PRIORITY APPLN. INFO.: US 1990-488650 19900305

AB A device useful for the **treatment** of infections of the middle ear in a **prophylactic** manner uses a biodegradable

support incorporating a **therapeutically** active agent, such as a drug. The device can be surgically inserted into the middle ear and there expand in order to substantially contact the walls of the middle ear. As it biodegrades, the expanded device provides prolonged, responsive release of active agent to the middle ear. The support is a polyester amide, poly(glycolic acid), poly(lactic acid), etc. A device was manufd. by coating a viscous suspension of poly-L-lactic acid and ampicillin onto a flat Teflon sheet. The resulting sheet was flexible enough to be rolled onto itself without cracking, but was strong and stiff enough to tend towards retaining its original flat shape. A coil device is also presented. The support preferably biodegrades in 2-18 mo. Biol. studies of the device are presented.

L12 ANSWER 8 OF 10 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1987:65325 CAPLUS
DOCUMENT NUMBER: 106:65325
TITLE: Experimental tympanosclerosis following **infection** with **Streptococcus pyogenes** and vitamin D3 intoxication
AUTHOR(S): Mann, W.
CORPORATE SOURCE: Sch. Med., Univ. Freiburg, Freiburg/Br., Fed. Rep. Ger.
SOURCE: Arch. Oto-Rhino-Laryngol. (1986), 243(5), 296-303
CODEN: AORLCG; ISSN: 0302-9530
DOCUMENT TYPE: Journal
LANGUAGE: English
AB A rat animal model was used to study the ultrastructure of submucosal calcifications induced in the middle ear following inoculation with S. pyogenes and high doses of parenteral vitamin D3. The morphol. changes present in affected animals resembled the classical picture of tympanosclerosis. While calcification occurred around bacterial remnants and myelin structures, the most important calcification centers were lysosomal and nonlysosomal matrix vesicles in the extracellular spaces. These formed band-like calcifications close to the basal membrane without affecting the epithelial layer. This animal model offers the possibility of studying the effect of various **therapeutic** regimens in the **treatment** of the dynamic tympanosclerotic process.

L12 ANSWER 9 OF 10 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1970:448550 CAPLUS
DOCUMENT NUMBER: 73:48550
TITLE: Textile **collagen** [surgical] aid
INVENTOR(S): Krajicek, Milan; Chvapil, Milos; Dvorak, Jan
SOURCE: Czech., 3 pp.
CODEN: CZXXA9

DOCUMENT TYPE: Patent
LANGUAGE: Czech
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
CS 132737		19690615	CS	19670519

AB The usual polyester or poly(tetrafluoroethylene) artificial arteries are impregnated with **collagen** obtained from alkali swollen bovine glue stock. The **collagen** material contains 50-150 mg/g antibiotics or 0.3-0.5 mg/g staphylococcal and **streptococcal** phage lyzate to **prevent infection** after implantation. The resorbability of the **collagen** material by the tissue is controlled by hardening in a 1.5% aq. soln. of 2,4,6-trimethoxytriazine 10 min or by addn. of 1-3% glycerol based on the wt. of the material. The finished arteries are lined with heparin from a 1% aq. soln. for 2-4 hr. The **collagen** material penetrates the walls of the artery and **prevents** bleeding and infection.

L12 ANSWER 10 OF 10 CAPLUS COPYRIGHT 2001 ACS
ACCESSION NUMBER: 1960:112150 CAPLUS
DOCUMENT NUMBER: 54:112150
ORIGINAL REFERENCE NO.: 54:21448i,21449a-b
TITLE: The role and significance of hyaluronidase in dermatology
AUTHOR(S): Nastase, Gh.; Speranta, Gh.; Cahane, A.; Carniol, M.; Lazar, M.; Dobrescu, A.
SOURCE: Arch. klin. u. exptl. Dermatol. (1960), 211, 187-93
DOCUMENT TYPE: Journal
LANGUAGE: Unavailable

AB The roles of hyaluronidase (I) and antihyaluronidase (II) in 114 cases of skin ailments were studied. In 34 streptodermia cases, the level of I was directly proportional to the progress of the pathol. process, with titers of 1:825 to 1:25. The level of testicular II in 12 skin cancer cases was neg. in the testicles; in 10 of these cases, the presence of **streptococcus** II was observed, with titers of 1:128 to 1:8, which indicated that the **infection** aggravated the cancer process. In 44 cases with various types of syphilis, the presence of the diffusion-factor was investigated by detn. of II, according to the clotting technique; in all 44 cases, the titer of the testicular II was 1:120; in only 4 of these cases was testicular I found. The **treatment** of 12 cases of secondary keloid by local infiltration of I resulted in the healing of 4 and improvement in 2 cases; a discussion of the interrrelation of vitamin C, phosphatase, **collagen** formation, and Ca

fixation was given in this connection.

(FILE 'MEDLINE, BIOSIS, EMBASE, WPIDS, CONFSCI, SCISEARCH, JICST-EPLUS, JAPIO' ENTERED AT 10:21:08 ON 07 JUN 2001)

```
L8          334 SEA FILE=REGISTRY ABB=ON  PLU=ON  COLLAGEN ?/CN
L9        70333 SEA FILE=CAPLUS ABB=ON  PLU=ON  L8 OR COLLAGEN
L16        2783 SEA (TREAT? OR THERAP? OR PROPHYL? OR PREVENT?)(10A)((STR
                EPTOCOCC? OR PYOGENES OR GAS(5A) STREPTOCOCC?)(5A)
                INFECTION)
L18           2 SEA L9 AND L16


L8          334 SEA FILE=REGISTRY ABB=ON  PLU=ON  COLLAGEN ?/CN
L9        70333 SEA FILE=CAPLUS ABB=ON  PLU=ON  L8 OR COLLAGEN
L10          22 SEA FILE=CAPLUS ABB=ON  PLU=ON  L9 AND ((STREPTOCOCC? OR
                PYOGENES OR GAS(S)STREPTOCOCC?)(S)INFECTION)
L11          11 SEA FILE=CAPLUS ABB=ON  PLU=ON  L10 AND (TREAT? OR
                PROPHYL? OR THERAP? OR PREVENT?)
L13         110 SEA L11
L19          11 SEA L13 AND BIND?

=> s (l18 or l19) not l6
L20          12 (L18 OR L19) NOT L6

=> dup rem l20
PROCESSING COMPLETED FOR L20
L21          12 DUP REM L20 (0 DUPLICATES REMOVED)

L21  ANSWER 1 OF 12  WPIDS COPYRIGHT 2001   DERWENT INFORMATION LTD
ACCESSION NUMBER:     2000-442641 [38]   WPIDS
DOC. NO. CPI:         C2000-134725
TITLE:                New protein useful for preparation of vaccines for
                      treatment of strangles caused by
                      Streptococcus equi infection, is
                      able to bind to mammalian fibronectin.
DERWENT CLASS:        B04 C06 D16
INVENTOR(S):          FRYKBERG, L; GUSS, B; JACOBSSON, K; LINDMARK, H
PATENT ASSIGNEE(S):   (FRYK-I) FRYKBERG L; (GUSS-I) GUSS B; (JACO-I)
                      JACOBSSON K; (LIND-I) LINDMARK H
COUNTRY COUNT:        89
PATENT INFORMATION:

     PATENT NO    KIND  DATE      WEEK      LA   PG
     -------------------------------------------
     WO 2000037496 A1 20000629 (200038)* EN   32
        RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC
            MW NL OA PT SD SE SL SZ TZ UG ZW
         W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM
```

EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ
LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD
SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW
AU 2000030947 A 20000712 (200048)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2000037496 A1		WO 1999-SE2448	19991221
AU 2000030947 A		AU 2000-30947	19991221

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000030947 A		Based on	WO 200037496

PRIORITY APPLN. INFO: SE 1998-4491 19981222
AN 2000-442641 [38] WPIDS
AB WO 200037496 A UPAB: 20000811
NOVELTY - A protein (I), designated SFS, having an amino acid sequence encoded by a nucleic acid sequence (II) isolated from, and forms a portion of the genomes of Streptococcus equi, is new and can be expressed from (II) and **binds** specifically to mammalian fibronectin or its analog or fragment.
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:
(1) a DNA fragment (III) comprising (II);
(2) a recombinant DNA molecule (IV) comprising a replicable expression vector and (III);
(3) a host cell (V) comprising (III) or (IV);
(4) preparation of (I);
(5) a vaccine (VI) comprising (I);
(6) an polyclonal or monoclonal antibody (VII) specific for (I), or an antibody fragment;
(7) an antigenic preparation comprising an antigen consisting of (I);
(8) an antiserum comprising a polyclonal (VII); and
(9) preparation of an antiserum.
ACTIVITY - Antibacterial; antiinflammatory.
MECHANISM OF ACTION - Vaccine.
USE - (I), its analogs or fragments may be used for the preparation of a vaccine that protects horses against strangles caused by S. equi infection (claimed). The antibody and/or antiserum may also be used for the **prophylactic** or **therapeutic treatment** of S. equi infection in mammal, especially horses (claimed).

ADVANTAGE - The use of vaccines containing (I) provides a more effective protection against S. equi infections, with fewer side effects.
Dwg.0/4

L21 ANSWER 2 OF 12 MEDLINE
ACCESSION NUMBER: 2000270431 MEDLINE
DOCUMENT NUMBER: 20270431 PubMed ID: 10808095
TITLE: Adhesion of Streptococcus gallolyticus strains to extracellular matrix proteins.
AUTHOR: Vanrobaeys M; Haesebrouck F; Ducatelle R; De Herdt P
CORPORATE SOURCE: Department of Pathology, Bacteriology and Avian Diseases, Faculty of Veterinary Medicine, Ghent University, Salisburylaan 133, 9820, Merelbeke, Belgium.
SOURCE: VETERINARY MICROBIOLOGY, (2000 Jun 1) 74 (3) 273-80. Journal code: XBW; 7705469. ISSN: 0378-1135.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200007
ENTRY DATE: Entered STN: 20000714
Last Updated on STN: 20000714
Entered Medline: 20000706

AB Fourteen pigeon Streptococcus gallolyticus strains of differing virulence, were tested for their ability to adhere to immobilised fibronectin, **collagen** types I, III and IV. Eight, 2 and 13 strains were able to **bind** fibronectin, **collagen** types III and IV, respectively. None of the strains adhered to **collagen** type I. Heat **treatment**, proteolytic digestion or periodate **treatment** reduced the **binding** of S. gallolyticus to fibronectin and **collagen** type IV, suggesting that surface receptors contain proteins and carbohydrates. Although **binding** to these extracellular matrix proteins can play a role in the pathogenesis of streptococcosis in pigeons, **binding** properties could not be related to virulence, indicating that other factors determine differences in virulence among pigeon S. gallolyticus strains. Adhesion to **collagen** type IV may account in part for the distribution pattern of the lesions observed in naturally and experimentally infected pigeons.

L21 ANSWER 3 OF 12 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 1998081469 EMBASE
TITLE: Drug-induced linear IgA disease with antibodies to **collagen** VII.
AUTHOR: Wakelin S.H.; Allen J.; Zhou S.; Wojnarowska F.

CORPORATE SOURCE: Dr. S.H. Wakelin, St John's Institute of Dermatology, St Thomas' Hospital, London SE1 7EH, United Kingdom
SOURCE: British Journal of Dermatology, (1998) 138/2 (310-314).
Refs: 40
ISSN: 0007-0963 CODEN: BJDEAZ
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 013 Dermatology and Venereology
037 Drug Literature Index
038 Adverse Reactions Titles
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Linear IgA disease (LAD) is characterized by circulating and tissue-bound IgA antibodies against heterogeneous antigens in the cutaneous basement membrane zone. In most cases the cause is unknown, but a minority of cases has been drug induced. We report a 76-year-old man who developed an acute blistering eruption following high-dose penicillin **treatment** for pneumococcal septicaemia. Indirect immunofluorescence demonstrated dermal **binding** IgA antibodies, and Western blotting of serum showed reactivity with a 250 kDa dermal antigen corresponding to **collagen** VII of anchoring fibrils. Indirect immunoelectron microscopy showed antibody labelling in the lamina densa and sublamina densa zone. This is one of the few cases of drug-induced LAD in which the target antigen profile has been characterized, and the first in which the antigen has been shown to correspond to **collagen** VII.

L21 ANSWER 4 OF 12 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 96:703301 SCISEARCH
THE GENUINE ARTICLE: VH772
TITLE: ADHERENCE OF STREPTOCOCCUS-UBERIS TO BOVINE MAMMARY EPITHELIAL-CELLS AND TO EXTRACELLULAR-MATRIX PROTEINS
AUTHOR: ALMEIDA R A (Reprint); LUTHER D A; KUMAR S J; CALVINHO L F; BRONZE M S; OLIVER S P
CORPORATE SOURCE: UNIV TENNESSEE, INST AGR, DEPT ANIM SCI, KNOXVILLE, TN, 37901 (Reprint)
COUNTRY OF AUTHOR: USA
SOURCE: JOURNAL OF VETERINARY MEDICINE SERIES B-ZENTRALBLATT FUR VETERINARMEDIZIN REIHE B-INFECTIOUS DISEASES AND VETERINARY PUBLIC HEALTH, (SEP 1996) Vol. 43, No. 7, pp. 385-392.
ISSN: 0931-1793.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: AGRI
LANGUAGE: ENGLISH
REFERENCE COUNT: 29

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Adherence of an encapsulated (UT 101) and a non-encapsulated (UT 102) strain of **Streptococcus** uberis to a bovine mammary epithelial cell line (MAC-T) and to extracellular matrix proteins (ECMP) including fibronectin, **collagen** and laminin was investigated. S. uberis was co-cultured at 4 degrees C with MAC-T cell monolayers. Both strains of S. uberis adhered to MAC-T cells. However, the nonencapsulated strain of S. uberis adhered better to MAC-T cells than the encapsulated strain. Preincubation of MAC-T cells with lipoteichoic acid (LTA) and/or **treatment** of S. uberis with antibodies directed against the carboxyl-terminal half of type 24 M protein reduced adherence of both strains of S. uberis to MAC-T cells. Adherence to ECMP was measured by incubating bis-carboxyethyl-carboxyfluorescein acetomethyl ester (BCECF-AM) labelled S. uberis in 96-well plates coated with fibronectin, **collagen** or laminin. Both strains adhered to ECMP, however, the encapsulated strain adhered better to ECMP than the non-encapsulated strain. Results of this investigation demonstrated that both strains of S. uberis evaluated were capable of adhering to bovine mammary epithelial cells and to ECMP. Adherence of S. uberis to mammary epithelium may be an extremely important mechanism in the establishment and progression of bovine intramammary **infections**.

L21 ANSWER 5 OF 12 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 94:434038 SCISEARCH
THE GENUINE ARTICLE: NV809
TITLE: INTEGRIN-MEDIATED ADHESIVE PROPERTIES OF UROEPITHELIAL CELLS ARE INHIBITED BY **TREATMENT** WITH BACTERIAL TOXINS
AUTHOR: ELGAVISH A (Reprint); PATTANAIK A; LLOYD K; REED R
CORPORATE SOURCE: UNIV ALABAMA, SCH MED, DEPT COMPARAT MED, BIRMINGHAM, AL, 35294 (Reprint); UNIV ALABAMA, SCH MED, DIV UROL, BIRMINGHAM, AL, 35294
COUNTRY OF AUTHOR: USA
SOURCE: AMERICAN JOURNAL OF PHYSIOLOGY, (JUN 1994) Vol. 266, No. 6, Part 1, pp. C1552-C1559.
ISSN: 0002-9513.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 53

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Gram-negative bacteria are a dominant cause of urinary tract **infection**, and their ability to produce toxins is an important virulence attribute. Cellular mechanisms triggered by the production of toxins in the lower urinary tract have not been completely defined. Ureteral epithelial cells (UT; A. Elgavish,

Infect. Immun. 61: 3304-3312, 1993) have served as an in vitro model to explore the possibility that bacterial toxins act on UT by affecting integrin-mediated adhesive properties. The effect of **treatment** with lipopolysaccharides (LPS) from three strains of the gram-negative Escherichia coli [055:B5 (LPS-1), 0111:B4 (LPS-4), and 0127:B8 (LPS-5)] and lipoteichoic acids from two gram-positive bacteria, **Streptococcus** faecalis (LT-2) and Bacillus subtilis (LT-3), were examined. LPS-5 inhibited markedly UT attachment to **collagen** and fibronectin. LPS-4 had no effect, whereas LPS-1 inhibited UT attachment to **collagen** but not to fibronectin. The fact that LPS-5 and LT-2 inhibited an Arg-Gly-Asp sequence-sensitive component of UT attachment to fibronectin is consistent with the possibility that these toxins acted via a mechanism involving typical fibronectin receptors. UT spreading was inhibited markedly by LPS-1, LT-2, and LT-3, whereas LPS-4 and LPS-5 had no effect. Because clustering of integrins is a crucial step in integrin-mediated signal transduction, the possibility that toxins inhibited spreading by affecting clustering was tested. **Treatment** with LT-2, which inhibited spreading dramatically, abolished completely a UT cell population containing more than five to eight beta(1)- or beta(4)-subunit-containing integrin clusters. Moreover, the cell population displaying low numbers of beta(1)-clusters per cell decreased considerably. LPS-5, which had no effect on spreading, did not affect clustering of beta(1)- or beta(4)-subunit-containing integrins. Taken together, the present studies are consistent with the possibility that **treatment** with certain bacterial toxins inhibits UT attachment and spreading on **collagen** and fibronectin and that integrins are involved in their mechanism of action.

L21 ANSWER 6 OF 12 MEDLINE
ACCESSION NUMBER: 92112291 MEDLINE
DOCUMENT NUMBER: 92112291 PubMed ID: 1530927
TITLE: Induction of a putative laminin-**binding** protein of Streptococcus gordonii in human infective endocarditis.
AUTHOR: Sommer P; Gleyzal C; Guerret S; Etienne J; Grimaud J A
CORPORATE SOURCE: Department of Pathology, Centre National de la Recherche Scientifique URA 1459, Institut Pasteur of Lyon, France.
SOURCE: INFECTION AND IMMUNITY, (1992 Feb) 60 (2) 360-5. Journal code: GO7; 0246127. ISSN: 0019-9567.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199202

ENTRY DATE: Entered STN: 19920308
Last Updated on STN: 19920308
Entered Medline: 19920218

AB There is evidence to suggest that the virulence of Streptococcus strains in infective endocarditis might be due to the expression of **binding** sites for the extracellular matrix proteins of damaged valves. In this communication, we draw attention to one laminin-**binding** protein from a strain of Streptococcus gordonii isolated from a patient with human endocarditis. This 145-kDa protein was found on the cell wall of the bacterium. The level of expression of this **binding** protein might be regulated by the presence of extracellular matrix proteins: the protein was lacking after in vitro selection of laminin, **collagen** I, and fibronectin nonbinding variants, and it was recovered after growth of the variants when laminin or **collagen** I was added to the growth medium. It was also missing after 10 subcultures in minimal medium, indicating some positive control. Furthermore, the 145-kDa protein was recognized as a major antigen by sera from patients **treated** for streptococcal infective endocarditis, while sera from patients with valvulopathies gave only slight recognition, suggesting an increase of the expression of this protein during infective endocarditis. It was also shown that the 145-kDa protein carried a **collagen** I-like determinant detected with anti-human **collagen** I antibodies.

L21 ANSWER 7 OF 12 SCISEARCH COPYRIGHT 2001 ISI (R)
ACCESSION NUMBER: 92:689979 SCISEARCH
THE GENUINE ARTICLE: JZ610
TITLE: COMPARATIVE-STUDIES ON **BINDING** OF VITRONECTIN AND FIBRONECTIN TO GROUP-A AND GROUP-C STREPTOCOCCI
AUTHOR: KOSTRZYNSKA M (Reprint); PAULSSON M; SCHMIDT K H; WADSTROM T
CORPORATE SOURCE: UNIV LUND, DEPT MED MICROBIOL, S-22362 LUND, SWEDEN; ACAD SCI GERMANY, CENT INST MICROBIOL & EXPTL THERAPY, JENA, GERMANY; NATL INST HYG, DEPT BACTERIOL, PL-00791 WARSAW, POLAND
COUNTRY OF AUTHOR: SWEDEN; GERMANY; POLAND
SOURCE: MICROBIOS, (1992) Vol. 71, No. 288-89, pp. 179-192. ISSN: 0026-2633.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: ENGLISH
REFERENCE COUNT: 26
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB **Binding** of I-125-labelled fibronectin and vitronectin to **streptococci** of group A (S. **pyogenes**), group

B (S. agalactiae) and group C (S. dysgalactiae and S. zooepidemicus) isolated from various human **infections** and bovine mastitis, and S. uberis bovine isolates, was studied. **Binding** of vitronectin and fibronectin was common among both human groups A and C, and bovine group C **streptococci**. S. agalactiae strains of human and bovine origin as well as S. uberis bovine isolates bound low levels of both proteins. The **binding** of radiolabelled fibronectin and vitronectin to selected groups A and C **streptococcal** strains was specific, time-dependent and occurred with both live and heat-killed (80-degrees-C for 1 5 min) cells. **Binding** declined rapidly after **treatment** of cells with trypsin or proteinase K, while pepsin digestion at pH 5.5 affected vitronectin but not fibronectin **binding**.

L21 ANSWER 8 OF 12 MEDLINE
ACCESSION NUMBER: 89290367 MEDLINE
DOCUMENT NUMBER: 89290367 PubMed ID: 2661319
TITLE: Specific **binding** of **collagen** type IV to Streptococcus pyogenes.
AUTHOR: Kostrzynska M; Schalen C; Wadstrom T
CORPORATE SOURCE: National Institute of Hygiene, Department of Bacteriology, Warsaw, Poland.
SOURCE: FEMS MICROBIOLOGY LETTERS, (1989 May) 50 (1-2) 229-33.
Journal code: FML; 7705721. ISSN: 0378-1097.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198908
ENTRY DATE: Entered STN: 19900309
Last Updated on STN: 19900309
Entered Medline: 19890809

AB Many strains of **Streptococcus pyogenes** are capable of **binding** type IV **collagen**. In the present study, all 50 S. **pyogenes** strains isolated from patients with acute glomerulonephritis showed high or moderate affinity for radiolabelled type IV **collagen**. A majority of strains of other sources, such as reference strains of various M-types and strains isolated from patients with pharyngeal **infections** also bound type IV **collagen**; however, a number of weak **binders** or non-**binders** were found among those. The **collagen** type IV **binding** component(s) on S. **pyogenes** were susceptible to proteinase K digestion, partially sensitive to trypsin but insensitive to pepsin **treatment** at pH 5.5. According to tests with three M-positive strains and their M-negative derivatives, the

binding was not dependent on M-protein. The **binding** was saturable with time and inhibited by unlabelled type IV **collagen**. Partially inhibition was found with type II **collagen**, gelatin and fibrinogen but not with a number of other serum proteins.

L21 ANSWER 9 OF 12 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 89132201 EMBASE
DOCUMENT NUMBER: 1989132201
TITLE: Specific **binding** of **collagen** type IV to Streptococcus pyogenes.
AUTHOR: Kostrzynska M.; Schalen C.; Wadstrom T.
CORPORATE SOURCE: National Institute of Hygiene, Department of Bacteriology, Warsaw, Poland
SOURCE: FEMS Microbiology Letters, (1989) 59/1-2 (229-233). ISSN: 0378-1097 CODEN: FMLED7
COUNTRY: Netherlands
DOCUMENT TYPE: Journal
FILE SEGMENT: 004 Microbiology
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Many strains of **Streptococcus pyogenes** are capable of **binding** type IV **collagen**. In the present study, all 50 S. **pyogenes** strains isolated from patients with acute glomerulonephritis showed high or moderate affinity for radiolabelled type IV **collagen**. A majority of strains of other sources, such as reference strains of various M-types and strains isolated from patients with pharyngeal **infections** also bound type IV **collagen**; however, a number of weak **binders** or non-**binders** were found among those. The collage type IV **binding** component(s) on S. **pyogenes** were susceptible to proteinase K digestion, partially sensitive to trypsin but insensitive to pepsin **treatment** at pH 5.5. According to tests with three M-positive strains and their M-negative derivatives, the **binding** was not dependent on M-protein. The **binding** was saturable with time and inhibited by unlabelled type IV collage. Partially inhibition was found with type II **collagen**, gelatin and fibrinogen but not with a number of other serum proteins.

L21 ANSWER 10 OF 12 MEDLINE
ACCESSION NUMBER: 89180958 MEDLINE
DOCUMENT NUMBER: 89180958 PubMed ID: 3333806
TITLE: **Binding** of fibronectin, fibrinogen and type II **collagen** to streptococci isolated from bovine mastitis.
AUTHOR: Mamo W; Froman G; Sundas A; Wadstrom T

CORPORATE SOURCE: Department of Veterinary Microbiology, Swedish University of Agricultural Sciences, Uppsala.
SOURCE: MICROBIAL PATHOGENESIS, (1987 Jun) 2 (6) 417-24.
Journal code: MIC; 8606191. ISSN: 0882-4010.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198904
ENTRY DATE: Entered STN: 19900306
Last Updated on STN: 19970203
Entered Medline: 19890427

AB **Binding** of 125I-labelled fibronectin, fibrinogen and type II **collagen** to group B (S. agalactiae), group C (S. dysgalactiae and S zooepidemicus), group E (S. uberis) and nontypable streptococci isolated from bovine mastitis was studied. S. agalactiae and S. uberis were found to **bind** low levels of all three proteins, while S. zooepidemicus bound high levels. **Binding** of the proteins to S. dysgalactiae varied, i.e. fibronectin was high, fibrinogen moderate and **collagen** low. Nontypable strains showed moderate or low **binding** of all proteins. Both hydrophobic and hydrophilic strains were found to **bind** fibronectin. For S. dysgalactiae the specific fibronectin **binding** ranged from 70% to 10% and for S. zooepidemicus it was more than 80% and this **binding** was sensitive to papain **treatment**. The **binding** of 29K-fibronectin fragment to one S. dysgalactiae strain showed an affinity of KD = 2.6 x 10(-8) M and the number of **binding** sites per colony forming unit (CFU) was calculated at 11,000.

L21 ANSWER 11 OF 12 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1985:384860 BIOSIS
DOCUMENT NUMBER: BA80:54852
TITLE: STUDY OF THE ETIOLOGICAL FACTORS AND DEVELOPMENTAL PARTICULARITIES OF CHRONIC RENAL INSUFFICIENCY IN 1275 CASES.
AUTHOR(S): BULIGESCU L; PROCA E; COSTANDACHE M; MUNTEANU V; SERBANESCU M; BUBENEK S; ZIDARESCU C; POPESCU A; POPESCU F
CORPORATE SOURCE: CLIN. MED., CLIN. CHIRURGIE, UROLOGICA, SPITALUL CLIN., FUNDENI, BUCURESTI.
SOURCE: REV MED INTERNA NEUROL PSIHIATR NEUROCHIR DERMATO-VENEROL SER MED INTERNA, (1984 (RECD 1985)) 36 (5), 443-456.
CODEN: RMIIDY. ISSN: 0303-8424.
FILE SEGMENT: BA; OLD
LANGUAGE: Romanian
AB The causes and development of 1275 cases of chronic renal

insufficiency (CRI) hospitalized during the last 5 yr were studied. The results showed the high proportion of CRI in the internal medicine departments (7.5% in the Fundeni Medical Clinic) and the high mortality rate of these cases (25.8% of the total number of deaths). Etiologic particularities in the group of the prevalence of glomerulonephritis and interstitial nephropathies, which was greater, in women than in Balkanic nephropathy. The etiologic spectrum differed according to age and sex with the predominance in youths streptococcal glomerulo-nephritis, the primary glomerulo-nephritis nephrotic form and other renal malformations, apart from polycystic kidney, without significance between the sexes. After the age of 30, in males, glomerulonephritis is a significant increased when compared to diabetic vascular and Balkanic nephropathies. In women nephropathies in **collagen** diseases and in exclusively nephropathies. The development of CRI is influenced by the etiologic substrate, being more rapid and severe in glomerulonephritis and more benign in interstitial nephropathies and polycystic kidney. The study contributes to knowledge of the etiologic spectrum of CRI in Romania, helping in the orientation of prophylactic measures in glomerular and interstitial affections with a view to diminishing the morbidity.

L21 ANSWER 12 OF 12 BIOSIS COPYRIGHT 2001 BIOSIS
ACCESSION NUMBER: 1977:211017 BIOSIS
DOCUMENT NUMBER: BA64:33381
TITLE: INTERACTION OF LIPO TEICHOIC-ACID OF GROUP A STREPTOCOCCI WITH HUMAN PLATELETS.
AUTHOR(S): BEACHEY E H; CHIANG T M; OFEK I; KANG A H
SOURCE: INFECT IMMUN, (1977) 16 (2), 649-654.
CODEN: INFIBR. ISSN: 0019-9567.
FILE SEGMENT: BA; OLD
LANGUAGE: Unavailable

AB The interaction of group A **streptococcal** lipoteichoic acid (LTA) [which may be associated with **streptococcal** pathogenicity or immunotoxic reactions during **streptococcal infections**] with mammalian cell membranes was studied in human platelets. The **binding** of LTA to platelets was platelet concentration and time dependent. **Binding** approached a maximum within 10 min of incubation. The bound LTA could be displaced by adding a 50-fold excess of unlabeled LTA. An association constant of 1.9 .times. 10-7 M was calculated, and only 1 population of **binding** sites was detected. Immunoferritin labeling of LTA-**treated** platelets demonstrated a patchy distribution of LTA **binding** sites on the platelet surface. LTA inhibited **collagen**- and .alpha.1 chain-induced platelet aggregation, but not the platelet release reaction, suggesting that the LTA and **collagen binding** sites on human platelets are distinct. Apparently, LTA **binds**

to platelets and interferes with **collagen**-induced aggregation although **collagen** is still able to attach to **binding** sites to trigger the release reaction.

(FILE 'MEDLINE' ENTERED AT 10:28:44 ON 07 JUN 2001)

L22 48694 SEA FILE=MEDLINE ABB=ON PLU=ON COLLAGEN/CT
L23 17001 SEA FILE=MEDLINE ABB=ON PLU=ON "STREPTOCOCCAL INFECTION S"/CT
L24 18 SEA FILE=MEDLINE ABB=ON PLU=ON L22 AND L23

L24 ANSWER 1 OF 18 MEDLINE
AN 2001043858 MEDLINE
TI Sporadic case of X-chromosomal Alport syndrome in a consanguineous family.
AU Ermisch B; Gross O; Netzer K O; Weber M; Brandis M; Zimmerhackl L B
SO PEDIATRIC NEPHROLOGY, (2000 Aug) 14 (8-9) 758-61.
Journal code: AVR. ISSN: 0931-041X.
AB Alport syndrome (AS) is a genetic disorder of basement membranes caused by mutations in type IV collagen genes that is characterized by chronic hematuria and progressive nephropathy leading to renal failure. The main extrarenal features include sensorineural hearing loss and ocular lesions. The mode of inheritance is X-linked dominant in about 80%-85% of the affected families, whereas autosomal transmission is rarely encountered. We report a male patient originating from a healthy consanguineous Lebanese family who presented with an unusual association of obstructive uropathy and AS. Hematuria and proteinuria were initially attributed to a suspected poststreptococcal glomerulonephritis (GN) and high-grade subpelvic ureteral stenosis. Persistence of symptoms after medical treatment of poststreptococcal GN and surgical correction of obstructive uropathy finally led to renal biopsy. The observed ultrastructural changes of the glomerular basement membrane were typical for AS. Molecular genetic studies revealed a previously undescribed de novo mutation in the COL4A5 gene, excluding maternal heterozygotic carrier status. This case report emphasizes the importance of hereditary nephritis in the differential diagnosis of chronic hematuria, and demonstrates the value of molecular studies for genetic counselling in AS.

L24 ANSWER 2 OF 18 MEDLINE
AN 2000270431 MEDLINE
TI Adhesion of Streptococcus gallolyticus strains to extracellular matrix proteins.
AU Vanrobaeys M; Haesebrouck F; Ducatelle R; De Herdt P
SO VETERINARY MICROBIOLOGY, (2000 Jun 1) 74 (3) 273-80.
Journal code: XBW; 7705469. ISSN: 0378-1135.
AB Fourteen pigeon Streptococcus gallolyticus strains of differing virulence, were tested for their ability to adhere to immobilised

fibronectin, collagen types I, III and IV. Eight, 2 and 13 strains were able to bind fibronectin, collagen types III and IV, respectively. None of the strains adhered to collagen type I. Heat treatment, proteolytic digestion or periodate treatment reduced the binding of S. gallolyticus to fibronectin and collagen type IV, suggesting that surface receptors contain proteins and carbohydrates. Although binding to these extracellular matrix proteins can play a role in the pathogenesis of streptococcosis in pigeons, binding properties could not be related to virulence, indicating that other factors determine differences in virulence among pigeon S. gallolyticus strains. Adhesion to collagen type IV may account in part for the distribution pattern of the lesions observed in naturally and experimentally infected pigeons.

L24 ANSWER 3 OF 18 MEDLINE
AN 1999381588 MEDLINE
TI Immunohistochemical distribution of extracellular matrix components and keratin in experimentally induced otitis media.
AU Harada T; Juhn S K; Kim Y; Sakakura Y
SO ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY, (1999 Aug) 108 (8) 769-76.
Journal code: 5Q2; 0407300. ISSN: 0003-4894.
AB The distribution of collagen types I, III, and IV and of laminin, fibronectin, and keratin was studied in otitis media experimentally induced by Streptococcus pneumoniae in the chinchilla. The expression of interstitial collagen types I and III and of fibronectin was increased in the subepithelial space that was thickened by inflammation in the acute period of infection. The expression of collagen type IV in the subepithelial space could be seen in the early period. The epithelial cells in the middle ear changed from flat cuboidal to pseudostratified columnar in pneumococcus-inoculated ears, and the number of keratin-positive epithelial cells in the middle ear increased remarkably after infection. These results indicate that changes in epithelial cell differentiation effected by the extracellular matrix correlate with changes in expression of keratin. It is proposed that the extracellular matrix may contribute to tissue repair in the middle ear after bacterial infection by interfering with cell proliferation of epithelial cells and fibroblasts.

L24 ANSWER 4 OF 18 MEDLINE
AN 1999242824 MEDLINE
TI SFS, a novel fibronectin-binding protein from Streptococcus equi, inhibits the binding between fibronectin and collagen.
AU Lindmark H; Guss B
SO INFECTION AND IMMUNITY, (1999 May) 67 (5) 2383-8.
Journal code: GO7; 0246127. ISSN: 0019-9567.
AB The obligate parasitic bacterium Streptococcus equi subsp. equi is

the causative agent of strangles, a serious disease of the upper respiratory tract in horses. In this study we have, using shotgun phage display, cloned from S. equi subsp. equi and characterized a gene, called sfs, encoding a protein termed SFS, representing a new type of fibronectin (Fn)-binding protein. The sfs gene was found to be present in all 50 isolates of S. equi subsp. equi tested and in 41 of 48 S. equi subsp. zooepidemicus isolates tested. The sfs gene is down-regulated during growth in vitro compared to fnz, a previously characterized gene encoding an Fn-binding protein from S. equi subsp. zooepidemicus. Sequence comparisons revealed no similarities to previously characterized Fn-binding proteins, but high scores were obtained against collagen. Besides similarity due to the high content of glycine, serine, and proline residues present in both proteins, there was a nine-residue motif present both in collagen and in the Fn-binding domain of SFS. By searching the Oklahoma S. pyogenes database, we found that this motif is also present in a potential cell surface protein from S. pyogenes. Protein SFS was found to inhibit the binding between Fn and collagen in a concentration-dependent way.

L24 ANSWER 5 OF 18 MEDLINE
AN 1998053959 MEDLINE
TI Invasion of dentinal tubules by oral streptococci is associated with collagen recognition mediated by the antigen I/II family of polypeptides.
AU Love R M; McMillan M D; Jenkinson H F
SO INFECTION AND IMMUNITY, (1997 Dec) 65 (12) 5157-64.
Journal code: GO7; 0246127. ISSN: 0019-9567.
AB Cell surface proteins SspA and SspB in Streptococcus gordonii and SpaP in Streptococcus mutans are members of the antigen I/II family of polypeptides produced by oral streptococci. These proteins are adhesins and mediate species-specific binding of cells to a variety of host and bacterial receptors. Here we show that antigen I/II polypeptides are involved in the attachment of oral streptococci to collagen and that they also determine the ability of these bacteria to invade human root dentinal tubules. Wild-type S. gordonii DL1 (Challis) cells showed heavy invasion of tubules to a depth of approximately 200 microm, whereas the abilities of cells of isogenic mutant strains OB220 (sspA) and OB219 (sspA sspB) to invade were 50 and >90% reduced, respectively. Likewise, wild-type S. mutans NG8 cells invaded dentinal tubules, whereas cells of isogenic mutant strain 834 (spaP) did not. The invasive abilities of strains OB220 and OB219 were restored by heterologous expression of S. mutans SpaP polypeptide in these strains. The extents of tubule invasion by various wild-type and mutant strains correlated with their levels of adhesion to type I collagen, a major component of dentin. Furthermore, S. gordonii DL1 cells exhibited a growth response to collagen by forming long chains. This was not shown by ssp mutants

but was restored by the expression of SpaP in these cells. The production of SspA polypeptide by S. gordonii DL1, but not production of SspB polypeptide by strain OB220 (sspA), was enhanced in the presence of collagen. These results are the first to demonstrate that antigen I/II family polypeptides bind collagen and mediate a morphological growth response of streptococci to collagen. These antigen I/II polypeptide activities are critical for intratubular growth of streptococci and thus for establishment of endodontic infections.

L24 ANSWER 6 OF 18 MEDLINE
AN 97066398 MEDLINE
TI Platelet-streptococcal interactions in endocarditis.
AU Herzberg M C
SO CRITICAL REVIEWS IN ORAL BIOLOGY AND MEDICINE, (1996) 7 (3) 222-36.
Ref: 117
Journal code: A41; 9009999. ISSN: 1045-4411.
AB Infective endocarditis is characterized by the formation of septic masses of platelets on the surfaces of heart valves and is most commonly caused by viridans streptococci. Streptococcal virulence in endocarditis involves factors that promote infectivity and pathogenicity. Adhesins and exopolysaccharide (glycocalyx) contribute to infectivity. Although many factors may contribute to pathogenicity, the platelet aggregation-associated protein (PAAP) of Streptococcus sanguis contributes directly to the development of experimental endocarditis. PAAP is synthesized as a rhamnose-rich glycoprotein of 115 kDa and contains a collagen-like platelet-interactive domain, pro-gly-glu-gln-gly-pro-lys. Expressed on the cell wall of platelet aggregation-inducing strains (Agg+) of S. sanguis, PAAP apparently interacts with a signal-transducing receptor complex on platelets, which includes a novel 175-kDa alpha 2-integrin-associated protein and a 65-kDa collagen-binding component. From available data, the role of PAAP in the pathogenesis of experimental endocarditis may be explained by a proposed mechanistic model. On injured heart valves, PAAP first enhances platelet accumulation into a fibrin-enmeshed thrombus (vegetation), within which S. sanguis colonizes. Colonizing bacteria must resist platelet microbicidal protein (PMPR). The aggregation of platelets on the heart valve may be potentiated by an ectoATPase expressed on the surface of the S. sanguis and platelet alpha-adrenoreceptors that respond to endogenous catecholamines. The expression of PAAP may be modified during infection. Collagen is exposed on damaged heart valves; fever (heat shock) occurs during endocarditis. In response to heat shock or collagen in vitro, PAAP expression is altered. After colonization, streptococcal exotoxin(s) may cause fever. Proteases and other enzymes from streptococci and host sources may directly destroy the heart valves. When PAAP is unexpressed or neutralized with specific antibodies, experimental

endocarditis runs a milder course and vegetations are smaller. The data suggest strongly, therefore, that the role of PAAP may overlap the colonization function of putative adhesins such as FimA or SsaB. Finally, PAAP also contributes to the development of the characteristic septic mural thrombus (vegetation) of infective endocarditis and the signs of valvular pathology.

L24 ANSWER 7 OF 18 MEDLINE
AN 96001605 MEDLINE
TI Salivary specific antibodies in relation to adhesion of Streptococcus pyogenes to pharyngeal cells of patients with rheumatic fever and rheumatic heart disease.
AU Kumar K S; Ganguly N K; Chandrashekher Y; Anand I S; Wahi P L
SO ADVANCES IN EXPERIMENTAL MEDICINE AND BIOLOGY, (1995) 371A 677-9. Journal code: 2LU; 0121103. ISSN: 0065-2598.

L24 ANSWER 8 OF 18 MEDLINE
AN 95302969 MEDLINE
TI Group B streptococci adhere to a variant of fibronectin attached to a solid phase.
AU Tamura G S; Rubens C E
SO MOLECULAR MICROBIOLOGY, (1995 Feb) 15 (3) 581-9. Journal code: MOM; 8712028. ISSN: 0950-382X.
AB Group B streptococci (GBS) are the leading cause of neonatal pneumonia and meningitis. Adherence of GBS to host tissues may play an important role in the pathogenesis of infection. The host molecules which mediate GBS adherence to host tissues are unknown. Many bacterial pathogens adhere to fibronectin, an important component of the extracellular matrix (ECM). Some pathogens adhere to both immobilized and soluble fibronectin, while others adhere to immobilized fibronectin, but not to soluble fibronectin. Previous data indicated that GBS do not adhere to soluble fibronectin. We studied the ability of GBS to adhere to immobilized fibronectin. Forty-five per cent of the input inoculum of COH1, a virulent GBS isolate, adhered to fibronectin immobilized on polystyrene. COH1 did not adhere to the other ECM proteins tested (laminin, type I collagen, vitronectin, and tenascin). Nine out of nine GBS strains from human sources tested adhered specifically to fibronectin at levels varying from 4-60%. We considered the possibility that GBS were adherent to a contaminant in the fibronectin preparation. Properties of fibronectin, including the presence of an immunologic epitope of fibronectin and binding to collagen, were verified to be properties of the molecule to which GBS adhere. COH1 adhered to fibronectin captured by a monoclonal antibody to fibronectin (FN-15), confirming that the molecule to which GBS adhere bears immunologic determinants of fibronectin. Adherence of COH1 to fibronectin was inhibited by collagen, confirming that the molecule to which GBS adhere binds to collagen. These data strongly suggest

that GBS adhere to fibronectin, and not to a contaminant.(ABSTRACT TRUNCATED AT 250 WORDS)

L24 ANSWER 9 OF 18 MEDLINE
AN 92393234 MEDLINE
TI Binding of laminin, type IV collagen, and vitronectin by Streptococcus pneumoniae.
AU Kostrzynska M; Wadstrom T
SO ZENTRALBLATT FUR BAKTERIOLOGIE, (1992 Jun) 277 (1) 80-3.
Journal code: BD7; 9203851. ISSN: 0934-8840.
AB Forty-three strains of Streptococcus pneumoniae were tested for their ability to bind radiolabelled laminin, collagen types I, II and IV, fibronectin, and vitronectin. Two basement membrane components, laminin and type IV collagen, interacted with many S. pneumoniae strains. All strains bound laminin and 28 (65%) bound collagen type IV. Approximately 60% of the strains bound vitronectin but only a few strains showed low binding of fibronectin and collagen type I and II.

L24 ANSWER 10 OF 18 MEDLINE
AN 92112291 MEDLINE
TI Induction of a putative laminin-binding protein of Streptococcus gordonii in human infective endocarditis.
AU Sommer P; Gleyzal C; Guerret S; Etienne J; Grimaud J A
SO INFECTION AND IMMUNITY, (1992 Feb) 60 (2) 360-5.
Journal code: GO7; 0246127. ISSN: 0019-9567.
AB There is evidence to suggest that the virulence of Streptococcus strains in infective endocarditis might be due to the expression of binding sites for the extracellular matrix proteins of damaged valves. In this communication, we draw attention to one laminin-binding protein from a strain of Streptococcus gordonii isolated from a patient with human endocarditis. This 145-kDa protein was found on the cell wall of the bacterium. The level of expression of this binding protein might be regulated by the presence of extracellular matrix proteins: the protein was lacking after in vitro selection of laminin, collagen I, and fibronectin nonbinding variants, and it was recovered after growth of the variants when laminin or collagen I was added to the growth medium. It was also missing after 10 subcultures in minimal medium, indicating some positive control. Furthermore, the 145-kDa protein was recognized as a major antigen by sera from patients treated for streptococcal infective endocarditis, while sera from patients with valvulopathies gave only slight recognition, suggesting an increase of the expression of this protein during infective endocarditis. It was also shown that the 145-kDa protein carried a collagen I-like determinant detected with anti-human collagen I antibodies.

L24 ANSWER 11 OF 18 MEDLINE

AN 89180958 MEDLINE
TI Binding of fibronectin, fibrinogen and type II collagen to streptococci isolated from bovine mastitis.
AU Mamo W; Froman G; Sundas A; Wadstrom T
SO MICROBIAL PATHOGENESIS, (1987 Jun) 2 (6) 417-24.
Journal code: MIC; 8606191. ISSN: 0882-4010.
AB Binding of 125I-labelled fibronectin, fibrinogen and type II collagen to group B (S. agalactiae), group C (S. dysgalactiae and S zooepidemicus), group E (S. uberis) and nontypable streptococci isolated from bovine mastitis was studied. S. agalactiae and S. uberis were found to bind low levels of all three proteins, while S. zooepidemicus bound high levels. Binding of the proteins to S. dysgalactiae varied, i.e. fibronectin was high, fibrinogen moderate and collagen low. Nontypable strains showed moderate or low binding of all proteins. Both hydrophobic and hydrophilic strains were found to bind fibronectin. For S. dysgalactiae the specific fibronectin binding ranged from 70% to 10% and for S. zooepidemicus it was more than 80% and this binding was sensitive to papain treatment. The binding of 29K-fibronectin fragment to one S. dysgalactiae strain showed an affinity of KD = 2.6 x 10(-8) M and the number of binding sites per colony forming unit (CFU) was calculated at 11,000.

L24 ANSWER 12 OF 18 MEDLINE
AN 87308332 MEDLINE
TI Nosocomial graft fragmentation and healing response of an ePTFE angioaccess graft.
AU Anderson J M; Hering T M; Ansel A L; Johnson J M
SO JOURNAL OF BIOMEDICAL MATERIALS RESEARCH, (1987 Aug) 21 (A2 Suppl) 153-62.
Journal code: HJJ; 0112726. ISSN: 0021-9304.
AB This investigation was directed toward the tissue reaction and wound healing response of an ePTFE prosthesis implanted in a human subject as an arteriovenous fistulae for over 7 years. Due to the frequent puncture of the prosthesis for hemodialysis access, the pattern of healing is markedly different from that normally observed in ePTFE grafts in humans. The ePTFE graft material of the AV fistula was completely incorporated in fibrous tissue with prominent pseudointima formation (inner capsule), fibrous tissue within the graft and a well-adhered periadventitial layer (outer capsule). In the portion of the graft most frequently punctured, the wall of the graft was composed mainly of fibrous tissue containing dissociated fragments of ePTFE. Biochemical analysis of the fibrous tissue across the wall of the graft revealed that it contained types I, III, and V collagen, with type I greater than III greater than V. The type V collagen was present in largest percentage at the luminal surface and in decreasing percentage in the ePTFE material and outer capsule. This analysis suggests that collagen type deposition in this prosthesis occurs in a manner similar to a normal healing

wound, except for the unusual pattern of type V collagen deposition, which may be an adaptive variation of the healing response.

L24 ANSWER 13 OF 18 MEDLINE
AN 87276820 MEDLINE
TI Does an intracervical infection influence the fibrinolytic activity and the collagen content of the fetal membranes? A study of ascending infections in pregnant ewes.
AU Evaldson G R; Larsson B; Jiborn H; Nord C E
SO EUROPEAN JOURNAL OF OBSTETRICS, GYNECOLOGY, AND REPRODUCTIVE BIOLOGY, (1987 Jul) 25 (3) 259-66.
Journal code: E4L; 0375672. ISSN: 0301-2115.
AB Apart from solely mechanical explanations, premature rupture of the membranes (PROM) has been suggested to be caused by an ascending infection. In order to investigate the role of infection in the mechanism of PROM, pregnant ewes were experimentally inoculated endocervically with either Bacteroides fragilis, Streptococcus intermedius or group B streptococci. These microorganisms were previously reported to be implicated in PROM in humans. The present investigation concerns the possible effect of an experimentally induced ascending infection on the collagen content and fibrinolytic activity (FA) of the fetal membranes. No relationship was observed between an ascending infection during pregnancy and the collagen content content of the fetal membrane specimens. It was concluded that changes in the collagen content bear no etiological significance in the mechanism of premature membrane rupture irrespective of an ascending infection's being present or not. Concerning FA in only one case, experiencing a Strept. intermedius amnionitis, was an elevated FA value observed. This finding indicates that the involvement of FA in the process of membrane rupture following ascending infection during pregnancy cannot be ruled out.

L24 ANSWER 14 OF 18 MEDLINE
AN 86142507 MEDLINE
TI Antibodies to basement membrane collagen and to laminin are present in sera from patients with poststreptococcal glomerulonephritis.
AU Kefalides N A; Pegg M T; Ohno N; Poon-King T; Zabriskie J; Fillit H
SO JOURNAL OF EXPERIMENTAL MEDICINE, (1986 Mar 1) 163 (3) 588-602.
Journal code: I2V; 2985109R. ISSN: 0022-1007.
AB Sera from patients with poststreptococcal glomerulonephritis (PSGN) known to have antibodies to proteoglycans were studied for the presence of antibodies against other basement membrane (BM) components. BM collagen (type IV) was isolated in the native state by extracting bovine anterior lens capsule (ALC) with 0.5 M acetic acid. The 7-S (collagenous) domain and the NC-1 (noncollagenous) domain of type IV collagen were obtained after bacterial collagenase digestion of ALC followed by gel filtration. Laminin was isolated

from the mouse EHS tumor and fibronectin from human plasma. Immunologic studies, using an ELISA and electroimmunoblot, revealed the presence of antibodies that reacted with intact, native type IV collagen and the 7-S collagenous domain of this molecule. Reaction with the NC-1 (noncollagenous) domain was minimal, and not higher than that obtained with control sera. Laminin reaction strongly with the patients' sera, but fibronectin did not. Unlike sera from patients with Goodpasture syndrome, which contain antibodies primarily against the NC-1 (noncollagenous) domain of type IV collagen, sera from patients with acute PSGN contain antibodies against all the major macromolecular components of BM. This difference in immunologic reactivity may account for the observed differences in the pathologic picture at the glomerular level.

L24 ANSWER 15 OF 18 MEDLINE
AN 75100919 MEDLINE
TI Periapical destructions caused by experimental pulpal inoculation of Streptococcus mutans in rats.
AU Rosengren L; Winblad B
SO ORAL SURGERY, ORAL MEDICINE, AND ORAL PATHOLOGY, (1975 Mar) 39 (3) 479-87.
Journal code: OJU; 0376406. ISSN: 0030-4220.
AB The development of pulpal and periapical changes in rat molars was studied after inoculation of Streptococcus mutans (GS-5) into the pulp chamber. Before injection into the pulp Streptococcus mutans was cultured on a special collagen substrate and "trained" to break down collagen. The destruction of the alveolar bone periapically could be demonstrated both roentgenologically and histopathologically. Large numbers of inflammatory cells in the pulp chamber and the periapical area, as well as carious dentin, were present. The pulpally inoculated bacteria could also be recovered from the systemic blood. The identity between the pulpally inoculated bacteria and the bacteria recovered from the blood was proved by gel precipitation.

L24 ANSWER 16 OF 18 MEDLINE
AN 74051200 MEDLINE
TI Ultrastructural and associated observations on clinical cases of mastitis in cattle.
AU Chandler R L; Reid I M
SO JOURNAL OF COMPARATIVE PATHOLOGY, (1973 Apr) 83 (2) 233-41.
Journal code: HVB; 0102444. ISSN: 0021-9975.

L24 ANSWER 17 OF 18 MEDLINE
AN 73234376 MEDLINE
TI Immunologic and histologic evaluation of the urinary bladder wall after group A streptococcal infection.
AU Harn S D; Keutel H J; Weaver R G

SO INVESTIGATIVE UROLOGY, (1973 Jul) 11 (1) 55-64.
Journal code: GWM; 0374747. ISSN: 0021-0005.

L24 ANSWER 18 OF 18 MEDLINE
AN 68243690 MEDLINE
TI Dental research: the past two decades. National Institute of Dental Research interdisciplinary programs have broadened the base of dental science.
AU Morris A L; Greulich R C
SO SCIENCE, (1968 Jun 7) 160 (832) 1081-8.
Journal code: UJ7; 0404511. ISSN: 0036-8075.

FILE 'HOME' ENTERED AT 10:29:34 ON 07 JUN 2001

09/494297

L5 ANSWER 1 OF 33 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1991:136702 BIOSIS
TI **ISOLATED DNA** REPEAT REGION FROM FCR-A-76 THE FC-BINDING PROTEIN GENE FROM AN M-TYPE 76 STRAIN OF **GROUP A STREPTOCOCCI** ENCODES A PROTEIN WITH FC-BINDING ACTIVITY.
AU HEATH D G; BOYLE M D P; CLEARY P P
CS DEP. ORAL BIOL., DENT. RES. INST., UNIV. MICH., ANN ARBOR, MICH. 48109-0402.
SO MOL MICROBIOL, (1990) 4 (12), 2071-2080.
CODEN: MOMIEE. ISSN: 0950-382X.
FS BA; OLD
LA English

L5 ANSWER 2 OF 33 CAPLUS COPYRIGHT 2001 ACS
AN 1991:96201 CAPLUS
DN 114:96201
TI **Isolated DNA** repeat region from fcrA76, the Fc-binding protein gene from an M-type 76 strain of **group A streptococci**, encodes a protein with Fc-binding activity
AU Heath, D. G.; Boyle, M. D. P.; Cleary, P. P.
CS Dep. Microbiol., Univ. Minnesota, Minneapolis, MN, 55455, USA
SO Mol. Microbiol. (1990), 4(12), 2071-9
CODEN: MOMIEE; ISSN: 0950-382X
DT Journal
LA English

L5 ANSWER 3 OF 33 EMBASE COPYRIGHT 2001 ELSEVIER SCI. B.V.
AN 91036995 EMBASE
DN 1991036995
TI **Isolated DNA** repeat region from fcrA76, the Fc-binding protein gene from an M-type 76 strain of **group A streptococci**, encodes a protein with Fc-binding activity.
AU Heath D.G.; Boyle M.D.P.; Cleary P.P.
CS Department of Oral Biology, Dental Research Institute, University of Michigan,Ann Arbor, MI 48109-0402, United States
SO Molecular Microbiology, (1990) 4/12 (2071-2079).
ISSN: 0950-382X CODEN: MOMIEE
CY United Kingdom
DT Journal; Article
FS 004 Microbiology
LA English
SL English

L5 ANSWER 4 OF 33 LIFESCI COPYRIGHT 2001 CSA
AN 90:73758 LIFESCI
TI **Isolated DNA** repeat region from fcrA76, the Fc-binding protein gene from an M-type 76 strain of **group A streptococci**, encodes a protein with Fc-binding activity.
AU Heath, D.G.; Boyle, M.D.P.; Cleary, P.P.
CS Dep. Oral Biol., Dent. Res. Inst., Univ. Michigan, Ann Arbor, MI 48109-0402, USA
SO MOL. MICROBIOL., (1990) vol. 4, no. 12, pp. 2071-2079.
DT Journal
FS J; L
LA English
SL English

L5 ANSWER 5 OF 33 MEDLINE
AN 91211616 MEDLINE
DN 91211616 PubMed ID: 2089220
TI **Isolated DNA** repeat region from fcrA76, the Fc-binding protein gene from an M-type 76 strain of **group A streptococci**, encodes a protein with Fc-binding activity.
AU Heath D G; Boyle M D; Cleary P P
CS Department of Microbiology, University of Minnesota, Minneapolis 55455.
NC A1-20445
SO MOLECULAR MICROBIOLOGY, (1990 Dec) 4 (12) 2071-9.

Journal code: MOM; 8712028. ISSN: 0950-382X.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199105
ED Entered STN: 19910616
Last Updated on STN: 19910616
Entered Medline: 19910529

L5 ANSWER 6 OF 33 SCISEARCH COPYRIGHT 2001 ISI (R)
AN 91:38564 SCISEARCH
GA The Genuine Article (R) Number: ER456
TI **ISOLATED DNA** REPEAT REGION FROM FCRA76, THE FC-BINDING PROTEIN GENE FROM AN M-TYPE 76 STRAIN OF **GROUP-A STREPTOCOCCI**, ENCODES A PROTEIN WITH FC-BINDING ACTIVITY
AU HEATH D G (Reprint); BOYLE M D P; CLEARY P P
CS UNIV MINNESOTA, DEPT BIOCHEM, MINNEAPOLIS, MN, 55455; VIRGINIA COMMONWEALTH UNIV, MED COLL VIRGINIA, DEPT MICROBIOL, RICHMOND, VA, 23298
CYA USA
SO MOLECULAR MICROBIOLOGY, (1990) Vol. 4, No. 12, pp. 2071-2079.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 39
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L5 ANSWER 7 OF 33 USPATFULL
AN 2001:152770 USPATFULL
TI Evolution of whole cells and organisms by recursive sequence recombination
IN delCardayre, Stephen, Belmont, CA, United States
Tobin, Matthew, San Jose, CA, United States
Stemmer, Willem P. C., Los Gatos, CA, United States
Ness, Jon E., Sunnyvale, CA, United States
Minshull, Jeremy, Menlo Park, CA, United States
Patten, Phillip, Menlo Park, CA, United States
Subramanian, Venkiteswaran, San Diego, CA, United States
Castle, Linda, Mountain View, CA, United States
Krebber, Claus M., Mountain View, CA, United States
Bass, Steven H., Hillsborough, CA, United States
PA Maxygen, Inc., Redwood City, CA, United States (U.S. corporation)
PI US 6287862 B1 20010911
AI US 2000-626410 20000726 (9)
RLI Division of Ser. No. US 1998-116188, filed on 15 Jul 1998
Continuation-in-part of Ser. No. WO 1998-US852, filed on 16 Jan 1998
PRAI US 1997-35054 19970107 (60)
DT Utility
FS GRANTED
LN.CNT 5146
INCL INCLM: 435/440.000
INCLS: 435/006.000; 536/023.100; 536/024.300; 935/076.000; 935/077.000; 935/078.000
NCL NCLM: 435/440.000
NCLS: 435/006.000; 536/023.100; 536/024.300; 935/076.000; 935/077.000; 935/078.000
IC [7]
ICM: C12N015-00
ICS: C12Q001-68; C07H021-02; C07H021-04
EXF 435/440; 435/6; 536/23.1; 536/24.3; 935/76; 935/77; 935/78
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 8 OF 33 USPATFULL
AN 2001:141887 USPATFULL
TI Non-IgA Fc binding forms of the group B **streptococcal** .beta. antigens
IN Tai, Joseph Y., Fort Washington, PA, United States
Blake, Milan S, Fulton, MD, United States
PA Baxter International Inc., Deerfield, IL, United States (U.S.

corporation)
PI US 6280738 B1 20010828
AI US 1997-923992 19970905 (8)
PRAI US 1996-24707 19960906 (60)
DT Utility
FS GRANTED
LN.CNT 1325
INCL INCLM: 424/197.110
INCLS: 424/190.100; 424/244.100; 424/282.100; 424/831.000; 514/054.000; 530/403.000; 530/825.000
NCL NCLM: 424/197.110
NCLS: 424/190.100; 424/244.100; 424/282.100; 424/831.000; 514/054.000; 530/403.000; 530/825.000
IC [7]
ICM: A61K039-09
ICS: A61K039-385; C07K014-315
EXF 424/190.1; 424/197.11; 424/244.1; 424/282.1; 424/831; 514/54; 530/350; 530/402; 530/403; 530/825
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 9 OF 33 USPATFULL
AN 2001:136775 USPATFULL
TI Compositions and methods for diagnosing and treating conditions, disorders, or diseases involving cell death
IN Lo, Donald C., Chapel Hill, NC, United States
Barney, Shawn, Apex, NC, United States
Thomas, Mary Beth, Chapel Hill, NC, United States
Portbury, Stuart D., Durham, NC, United States
Puranam, Kasturi, Durham, NC, United States
Katz, Lawrence C., Durham, NC, United States
PA Cogent Neuroscience, Inc., Durham, NC, United States (U.S. corporation)
PI US 6277974 B1 20010821
AI US 1999-461697 19991214 (9)
DT Utility
FS GRANTED
LN.CNT 4670
INCL INCLM: 536/023.100
INCLS: 536/023.100; 536/023.500; 424/093.200; 424/093.100; 424/093.210; 435/069.100; 435/325.000; 435/352.000; 435/320.100; 530/300.000; 530/350.000
NCL NCLM: 536/023.100
NCLS: 424/093.100; 424/093.200; 424/093.210; 435/069.100; 435/320.100; 435/325.000; 435/352.000; 530/300.000; 530/350.000; 536/023.500
IC [7]
ICM: C07H021-02
EXF 536/23.1; 536/23.4; 435/320.1; 435/325; 435/69.1; 530/300; 530/350; 424/93.2; 424/93.21; 424/93.1
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 10 OF 33 USPATFULL
AN 2001:134814 USPATFULL
TI Method for providing hyaluronic acid
IN Weigel, Paul H., Edmond, OK, United States
Papaconstantinou, John, Galveston, TX, United States
PA The Board of Regents of the University of Oklahoma, Norman, OK, United States (U.S. corporation)
PI US 37336 E1 20010821
US 5015577 19910514 (Original)
AI US 1999-281107 19990329 (9)
US 1989-401316 19890829 (Original)
DT Reissue
FS GRANTED
LN.CNT 1485
INCL INCLM: 435/101.000
INCLS: 435/252.300; 435/252.330; 435/253.400; 435/320.100; 435/849.000; 435/885.000; 435/193.000; 536/023.700
NCL NCLM: 435/101.000
NCLS: 435/193.000; 435/252.300; 435/252.330; 435/253.400; 435/320.100; 435/849.000; 435/885.000; 536/023.700

IC [7]
ICM: C12P019-04
ICS: C12N001-21; C12N009-10; C07H015-12
EXF 536/23.2; 536/23.7; 435/101; 435/193; 435/252.3; 435/252.33; 435/253.4; 435/320.1; 435/849; 435/885

L5 ANSWER 11 OF 33 USPATFULL
AN 2001:126017 USPATFULL
TI Erythromycins and process for their preparation
IN Leadlay, Peter Francis, Cambridge, United Kingdom
Staunton, James, Cambridge, United Kingdom
Cortes, Jesus, Cambridge, United Kingdom
Pacey, Michael Stephen, Broadstairs, United Kingdom
PA Biotica Technology Limited, Cambridge, United Kingdom (non-U.S. corporation)
Pfizer, Inc., New York, NY, United States (U.S. corporation)
PI US 6271255 B1 20010807
US 2001016598 A1 20010823
WO 9801571 19980115
AI US 1999-214454 19990916 (9)
WO 1997-GB1810 19970704
19990916 PCT 371 date
19990916 PCT 102(e) date
PRAI GB 1996-14189 19960705
GB 1997-10962 19970528
US 1996-24188 19960819 (60)
DT Utility
FS GRANTED
LN.CNT 2517
INCL INCLM: 514/450.000
INCLS: 549/271.000; 549/266.000; 549/029.000; 549/013.000; 536/007.200; 514/029.000
NCL NCLM: 514/450.000
NCLS: 514/029.000; 536/007.200; 549/013.000; 549/029.000; 549/266.000; 549/271.000
IC [7]
ICM: A61K031-365
ICS: C07D315-00
EXF 514/29; 514/450; 536/7.2; 549/286; 549/271; 549/13; 549/29
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 12 OF 33 USPATFULL
AN 2001:97699 USPATFULL
TI Evolution of whole cells and organisms by recursive sequence recombination
IN Tobin, Matthew, San Jose, CA, United States
Stemmer, William P. C., Los Gatos, CA, United States
Ness, Jon E., Sunnyvale, CA, United States
Minshull, Jeremy, Menlo Park, CA, United States
PA Maxygen, Inc., Redwood City, CA, United States (U.S. corporation)
PI US 6251674 B1 20010626
AI US 2000-499505 20000207 (9)
RLI Division of Ser. No. US 116188
PRAI US 1997-35054 19970107 (60)
DT Utility
FS GRANTED
LN.CNT 5013
INCL INCLM: 435/400.000
INCLS: 435/006.000; 536/023.100; 536/024.300; 935/076.000; 935/077.000; 935/078.000
NCL NCLM: 435/400.000
NCLS: 435/006.000; 536/023.100; 536/024.300
IC [7]
ICM: C12N015-00
ICS: C12Q001-68; C07H021-02; C07H021-04
EXF 435/440; 435/6; 435/91.2; 536/23.1; 536/24.3; 935/76; 935/77; 935/78
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 13 OF 33 USPATFULL

AN 2000:102088 USPATFULL
TI DNA encoding f.alpha.-2m-binding protein and protein encoded thereby
IN Guss, Bengt, Dag Hamarskjolds vag 238B, S-756 52 Uppsala, Sweden
Jonsson, Hans, Borjegatan 58C, S-752 29 Uppsala, Sweden
Lindberg, Martin, Kornvagen 5, S-752 57 Uppsala, Sweden
Mueller, Hans-Peter, Tjalinge 9, S-740 20 Brunna, Sweden
Rantamaki, Liisa K., Ojahaanpolku 6 B 20, FIN-016 00 Vantaa, Finland
PI US 6100055 20000808
WO 9507296 19950316
AI US 1996-669408 19960703 (8)
WO 1994-SE826 19940906
19960703 PCT 371 date
19960703 PCT 102(e) date
PRAI SE 1993-2855 19930906
DT Utility
FS Granted
LN.CNT 1659
INCL INCLM: 435/069.100
INCLS: 435/071.100; 435/071.200; 435/252.300; 435/254.110; 435/320.100; 435/471.000; 536/023.700; 530/350.000
NCL NCLM: 435/069.100
NCLS: 435/071.100; 435/071.200; 435/252.300; 435/254.110; 435/320.100; 435/471.000; 530/350.000; 536/023.700
IC [7]
ICM: C12N015-11
ICS: C12N015-63; C07K014-315
EXF 435/69.1; 435/252.3; 435/254.11; 435/320.1; 435/71.1; 435/71.2; 435/471; 435/325; 536/23.7; 935/58; 530/350
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 14 OF 33 USPATFULL
AN 2000:9689 USPATFULL
TI PCR identification and quantification of important Candida species
IN Jordan, Jeanne A., Pittsburgh, PA, United States
PA The University of Pittsburgh, Pittsburgh, PA, United States (U.S. corporation)
PI US 6017699 20000125
AI US 1996-624290 19960329 (8)
RLI Continuation-in-part of Ser. No. US 1995-491641, filed on 19 Jun 1995, now abandoned which is a continuation of Ser. No. US 1993-120780, filed on 15 Sep 1993, now patented, Pat. No. US 5426026
DT Utility
FS Granted
LN.CNT 1501
INCL INCLM: 435/006.000
INCLS: 536/024.310; 435/921.000
NCL NCLM: 435/006.000
NCLS: 435/921.000; 536/024.310
IC [6]
ICM: C12Q001-68
EXF 435/6; 435/921; 435/922; 435/923; 435/924; 536/24.31; 536/24.33
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 15 OF 33 USPATFULL
AN 1999:166795 USPATFULL
TI DNA sequence, related probes and primers for the detection of **Streptococcus** agalactiae
IN You, Qimin, Lutherville, MD, United States
PA Becton Dickinson and Company, Franklin Lakes, NJ, United States (U.S. corporation)
PI US 6004754 19991221
AI US 1998-10310 19980121 (9)
DT Utility
FS Granted
LN.CNT 1511
INCL INCLM: 435/006.000
INCLS: 435/091.100; 435/091.200; 536/023.100; 536/024.300; 536/024.320
NCL NCLM: 435/006.000
NCLS: 435/091.100; 435/091.200; 536/023.100; 536/024.300; 536/024.320

IC [6]
ICM: C12Q001-68
ICS: C12P019-34; C07H021-00; C07H021-02
EXF 435/6; 435/91.1; 435/91.2; 536/23.1; 536/24.3; 536/24.31; 536/24.32; 536/24.33
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 16 OF 33 USPATFULL
AN 1999:163420 USPATFULL
TI Species specific and universal DNA probes and amplification primers to rapidly detect and identify common bacterial pathogens and associated antibiotic resistance genes from clinical specimens for routine diagnosis in microbiology laboratories
IN Bergeron, Michel G., Sillery, Canada
Ouellette, Marc, Quebec, Canada
Roy, Paul H., Loretteville, Canada
PA Infectio Diagnostic, Inc., Canada (non-U.S. corporation)
PI US 6001564 19991214
AI US 1995-526840 19950911 (8)
RLI Continuation-in-part of Ser. No. US 1994-304732, filed on 12 Sep 1994
DT Utility
FS Granted
LN.CNT 5352
INCL INCLM: 435/006.000
INCLS: 435/091.200
NCL NCLM: 435/006.000
NCLS: 435/091.200
IC [6]
ICM: C12Q001-68
ICS: C12P019-34
EXF 435/6; 435/91.2; 536/22.1
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 17 OF 33 USPATFULL
AN 1999:155450 USPATFULL
TI Species-specific and universal DNA probes and amplification primers to rapidly detect and identify common bacterial pathogens and associated antibiotic resistance genes from clinical specimens for routine diagnosis in microbiology laboratories
IN Bergeron, Michel G., Sillery, Canada
Picard, Fran.cedilla.ois J., Ste-Foy, Canada
Ouellette, Marc, Quebec, Canada
Roy, Paul H., Loretteville, Canada
PA Infectio Diagnostic, Inc., Quebec, Canada (non-U.S. corporation)
PI US 5994066 19991130
AI US 1996-743637 19961104 (8)
RLI Continuation-in-part of Ser. No. US 1995-526840, filed on 11 Sep 1995
DT Utility
FS Granted
LN.CNT 8139
INCL INCLM: 435/006.000
INCLS: 435/091.200; 536/022.100
NCL NCLM: 435/006.000
NCLS: 435/091.200; 536/022.100
IC [6]
ICM: C12Q001-68
ICS: C12P019-34; C07H021-02
EXF 435/5; 435/6; 435/91.2; 536/22.1
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 18 OF 33 USPATFULL
AN 1999:124726 USPATFULL
TI Protein L and hybrid proteins thereof
IN Bjorck, Lars, Sodra Sandby, Sweden
Sjobring, Ulf, Lund, Sweden
PA Actinova Ltd., Lund, Sweden (non-U.S. corporation)
PI US 5965390 19991012
AI US 1997-795475 19970211 (8)
RLI Division of Ser. No. US 1994-325278, filed on 26 Oct 1994

PRAI SE 1992-1331 19920428
DT Utility
FS Granted
LN.CNT 1305
INCL INCLM: 435/069.100
INCLS: 435/252.300; 435/254.110; 435/320.100; 536/023.100
NCL NCLM: 435/069.100
NCLS: 435/252.300; 435/254.110; 435/320.100; 536/023.100
IC [6]
ICM: C12P021-02
ICS: C07H021-04; C12N001-21; C12N015-63
EXF 536/23.1; 536/24.1; 435/320.1; 435/69.1; 435/257.3; 435/252.31; 435/252.33; 435/254.11; 435/254.2; 435/254.21
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 19 OF 33 USPATFULL
AN 1999:113865 USPATFULL
TI Vaccines for nontypable haemophilus influenzae
IN Green, Bruce A., Pittsford, NY, United States
Zlotnick, Gary W., Penfield, NY, United States
PA Praxis Biologies, Inc., Rochester, NY, United States (U.S. corporation)
PI US 5955580 19990921
AI US 1995-449406 19950523 (8)
RLI Division of Ser. No. US 1990-491466, filed on 9 Mar 1990, now patented, Pat. No. US 5601831, issued on 11 Feb 1997 which is a continuation-in-part of Ser. No. US 1989-320971, filed on 9 Mar 1989, now abandoned
DT Utility
FS Granted
LN.CNT 1521
INCL INCLM: 530/350.000
INCLS: 424/256.100; 514/012.000
NCL NCLM: 530/350.000
NCLS: 424/256.100
IC [6]
ICM: C07K001-00
ICS: C07K014-285; A61K039-102
EXF 530/350; 424/256.1; 514/12
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 20 OF 33 USPATFULL
AN 1999:106576 USPATFULL
TI **Streptococcus** pneumoniae capsular polysaccharide genes and flanking regions
IN Yother, Janet, Birmingham, AL, United States
Dillard, Joseph, Hinsdale, IL, United States
PA UAB Research Foundation, Birmingham, AL, United States (U.S. corporation)
PI US 5948900 19990907
AI US 1997-867030 19970602 (8)
RLI Continuation-in-part of Ser. No. US 1994-243546, filed on 16 May 1994, now abandoned
DT Utility
FS Granted
LN.CNT 4586
INCL INCLM: 536/024.320
INCLS: 536/023.100; 536/023.700
NCL NCLM: 536/024.320
NCLS: 536/023.100; 536/023.700
IC [6]
ICM: C07H021-04
ICS: C07H021-02
EXF 536/23.1; 536/23.7; 536/24.32; 436/94; 435/91.1; 435/882; 435/6
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 21 OF 33 USPATFULL
AN 1999:75497 USPATFULL
TI **Streptococcus** suis adhesin protein and method for producing it
IN Tikkanen, Kaarina, Maaherrankatu 35 as 9 FIN-70100, Kuopio, Finland

Finne, Jukka, Katajanokanranta 3 A 5 FIN-00160, Helsinki, Finland
PI US 5919640 19990706
AI US 1997-889013 19970707 (8)
RLI Continuation-in-part of Ser. No. US 500895
PRAI FI 1993-413 19930129
DT Utility
FS Granted
LN.CNT 1038
INCL INCLM: 435/007.340
INCLS: 424/234.100; 435/006.000; 536/023.100; 536/024.320
NCL NCLM: 435/007.340
NCLS: 424/234.100; 435/006.000; 536/023.100; 536/024.320
IC [6]
ICM: G01N033-569
EXF 435/6; 536/23.1; 536/24.32; 424/234.1
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 22 OF 33 USPATFULL
AN 1998:82877 USPATFULL
TI Method for purification of protein "e" from haemophilus influenzae
IN Green, Bruce A., Pittsford, NY, United States
Zlotnick, Gary W., Penfield, NY, United States
PA Praxis Biologics, Inc., Rochester, NY, United States (U.S. corporation)
PI US 5780601 19980714
AI US 1995-447653 19950523 (8)
RLI Division of Ser. No. US 1990-491466, filed on 9 Mar 1990, now patented, Pat. No. US 5601831 which is a continuation-in-part of Ser. No. US 1989-320971, filed on 9 Mar 1989, now abandoned
DT Utility
FS Granted
LN.CNT 1503
INCL INCLM: 530/412.000
INCLS: 424/256.100; 530/350.000
NCL NCLM: 530/412.000
NCLS: 424/256.100; 530/350.000
IC [6]
ICM: C07K001-04
EXF 424/256.1; 530/300; 530/350; 530/412; 530/414; 530/415; 530/422
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 23 OF 33 USPATFULL
AN 97:117676 USPATFULL
TI Polyspecific immunoconjugates and antibody composites for targeting the multidrug resistant phenotype
IN Goldenberg, David M., Mendham, NJ, United States
PA Immunomedics, Inc., Morris Plains, NJ, United States (U.S. corporation)
PI US 5698178 19971216
AI US 1996-629387 19960408 (8)
RLI Division of Ser. No. US 1994-286430, filed on 5 Aug 1994
DT Utility
FS Granted
LN.CNT 2203
INCL INCLM: 424/001.490
INCLS: 424/009.341; 424/009.600; 424/001.530
NCL NCLM: 424/001.490
NCLS: 424/001.530; 424/009.341; 424/009.600
IC [6]
ICM: A61K049-00
EXF 424/9.341; 424/1.49; 424/9.6; 424/1.53
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 24 OF 33 USPATFULL
AN 97:104602 USPATFULL
TI Polyspecific immunoconjugates and antibody composites for targeting the multidrug resistant phenotype
IN Goldenberg, David M., Mendham, NJ, United States
PA Immunomedics, Inc., Morris Plains, NJ, United States (U.S. corporation)
PI US 5686578 19971111
AI US 1994-286430 19940805 (8)

DT Utility
FS Granted
LN.CNT 2133
INCL INCLM: 530/387.300
INCLS: 530/391.100; 530/391.900; 530/389.700; 530/389.500; 530/388.800; 530/388.850; 530/389.100; 530/388.200; 530/388.400
NCL NCLM: 530/387.300
NCLS: 530/388.200; 530/388.400; 530/388.800; 530/388.850; 530/389.100; 530/389.500; 530/389.700; 530/391.100; 530/391.900
IC [6]
ICM: C07K016-00
ICS: C07K016-18; C07K016-28; C07K016-12
EXF 530/387.3; 530/391.1-391.9; 530/389.7; 530/389.5; 530/388.8; 530/388.85; 530/389.1; 530/388.2; 530/388.4
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 25 OF 33 USPATFULL
AN 97:63922 USPATFULL
TI Lanthionine antibiotic compositions and methods
IN Caufield, Page W., Birmingham, AL, United States
Novak, Jan, Birmingham, AL, United States
PA University of Alabama at Birmingham Research Foundation, Birmingham, AL, United States (U.S. corporation)
PI US 5650320 19970722
AI US 1994-230473 19940420 (8)
DT Utility
FS Granted
LN.CNT 2836
INCL INCLM: 435/252.300
INCLS: 435/320.100; 435/242.330; 435/253.400; 536/023.700
NCL NCLM: 435/252.300
NCLS: 435/252.330; 435/253.400; 435/320.100; 536/023.700
IC [6]
ICM: C12N001-21
ICS: C12N015-63; C07H021-04
EXF 536/23.7; 435/320.1; 435/252.3
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 26 OF 33 USPATFULL
AN 97:12181 USPATFULL
TI Vaccines for nontypable Haemophilus influenzae
IN Green, Bruce A., Pittsford, NY, United States
Zlotnick, Gary W., Penfield, NY, United States
PA Praxis Biologics, Inc., Rochester, NY, United States (U.S. corporation)
PI US 5601831 19970211
AI US 1990-491466 19900309 (7)
RLI Continuation-in-part of Ser. No. US 1989-320971, filed on 9 Mar 1989, now abandoned
DT Utility
FS Granted
LN.CNT 1576
INCL INCLM: 424/256.100
INCLS: 424/282.100; 424/193.100; 424/192.100; 424/185.100
NCL NCLM: 424/256.100
NCLS: 424/185.100; 424/192.100; 424/193.100; 424/282.100
IC [6]
ICM: A61K039-102
ICS: A61K039-385
EXF 424/89; 424/92; 424/93; 424/256.1; 424/282.1; 424/193.1; 424/192.1; 424/185.1
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 27 OF 33 USPATFULL
AN 96:80142 USPATFULL
TI Polypeptides containing sequences characteristic of pyrrolidone carboxylyl peptidases, polynucleotides containing a sequence coding for such polypeptides, and their use, in particular for diagnostic purposes
IN Cleuziat, Philippe L., Lyons, France
Awade, Abalo, Rennes, France

Robert-Baudouy, Jeannine, Rillieux La Pape, France
Gayral, Jean-Pierre, Amberieu en Bugey, France
PA Bio Merieux, L'Etoile, France (non-U.S. corporation)
PI US 5552273 19960903
AI US 1993-107684 19931018 (8)
WO 1992-FR1237 19921223
19931018 PCT 371 date
19931018 PCT 102(e) date
PRAI FR 1991-16059 19911223
DT Utility
FS Granted
LN.CNT 1617
INCL INCLM: 435/006.000
INCLS: 435/069.100; 435/195.000; 435/227.000; 435/320.100; 435/240.200; 435/252.300; 530/387.100; 536/022.100; 536/023.100; 536/023.200; 536/023.700
NCL NCLM: 435/006.000
NCLS: 435/069.100; 435/195.000; 435/227.000; 435/252.300; 435/320.100; 530/387.100; 536/022.100; 536/023.100; 536/023.200; 536/023.700
IC [6]
ICM: C12Q001-68
ICS: C12P021-06; C12N001-20; C07H019-00
EXF 435/6; 435/69.1; 435/195; 435/227; 435/320.1; 435/240.2; 435/252.3; 530/387.1; 536/22.1; 536/23.1; 536/23.2; 536/23.7
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 28 OF 33 USPATFULL
AN 94:86318 USPATFULL
TI **Streptococcal** immunoglobulin a binding protein encoded by emmL2.2
IN Fischetti, Vincent A., West Hempstead, NY, United States
Bessen, Debra E., New York, NY, United States
PA Rockefeller University, New York, NY, United States (U.S. corporation)
PI US 5352588 19941004
AI US 1991-813584 19911224 (7)
DT Utility
FS Granted
LN.CNT 377
INCL INCLM: 435/069.100
INCLS: 530/350.000; 536/023.700; 435/320.100; 435/252.300; 435/252.330
NCL NCLM: 435/069.100
NCLS: 435/252.300; 435/252.330; 435/320.100; 530/350.000; 536/023.700
IC [5]
ICM: C07K013-00
ICS: C12N015-31; C12N001-21
EXF 536/23.7; 530/350; 435/252.3; 435/252.33; 435/320.1; 435/69.1
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 29 OF 33 USPATFULL
AN 94:60245 USPATFULL
TI Bacterial plasmin receptors as fibrinolytic agents
IN Boyle, Michael D. P., Whitehouse, OH, United States
Lottenberg, Richard, Gainesville, FL, United States
Broder, Christopher, Rockville, MD, United States
Von Mering, Gregory, Gainesville, FL, United States
PA University of Florida Research Foundation, Inc., Gainesville, FL, United States (U.S. corporation)
PI US 5328996 19940712
AI US 1992-928462 19920810 (7)
RLI Continuation-in-part of Ser. No. US 1990-524411, filed on 16 May 1990, now patented, Pat. No. US 5237050 which is a continuation-in-part of Ser. No. US 1989-330849, filed on 29 Mar 1989, now abandoned
DT Utility
FS Granted
LN.CNT 1522
INCL INCLM: 536/023.100
INCLS: 536/023.700; 424/094.640; 435/172.300; 530/350.000; 530/388.250; 530/381.000; 530/825.000
NCL NCLM: 536/023.100

NCLS: 424/094.640; 530/350.000; 530/381.000; 530/388.250; 530/825.000; 536/023.700
IC [5]
ICM: C07H017-00
EXF 536/23.1; 536/23.7
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 30 OF 33 USPATFULL
AN 94:57901 USPATFULL
TI Nucleic acid encoding N-acetylmuramidase M1
IN Lichenstein, Henri, Ventura, CA, United States
Langley, Keith, Newbury Park, CA, United States
Zukowski, Mark, Thousand Oaks, CA, United States
PA AMGEN Inc., Thousand Oaks, CA, United States (U.S. corporation)
PI US 5326858 19940705
AI US 1992-921371 19920728 (7)
RLI Continuation of Ser. No. US 1989-421820, filed on 16 Oct 1989, now abandoned
DT Utility
FS Granted
LN.CNT 913
INCL INCLM: 536/023.200
INCLS: 435/172.300; 435/206.000; 935/014.000; 424/094.610
NCL NCLM: 536/023.200
NCLS: 424/094.610; 435/206.000
IC [5]
ICM: C07H021-04
ICS: C12N015-56; A61K037-54
EXF 435/172.3; 435/206; 935/14; 536/23.2
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 31 OF 33 USPATFULL
AN 92:34054 USPATFULL
TI Protein G and/or fragments thereof
IN Bjorck, Lars, Sodra Sandby, Sweden
Kronvall, Goran, Lund, Sweden
Lindahl, Gunnar, Lund, Sweden
Kastern, William H., S.phi.borg, Denmark
PA Pharmacia LKB Biotechnology AB, Sweden (non-U.S. corporation)
PI US 5108894 19920428
AI US 1989-376160 19890705 (7)
RLI Continuation of Ser. No. US 1986-857764, filed on 30 Apr 1986, now abandoned
PRAI SE 1985-2162 19850503
DT Utility
FS Granted
LN.CNT 430
INCL INCLM: 435/006.000
INCLS: 435/007.320; 435/007.340; 435/069.100; 435/091.000; 435/172.300; 435/252.300; 435/252.330; 435/235.100; 435/320.100; 536/027.000; 530/350.000; 935/019.000; 935/029.000; 935/031.000; 935/041.000; 935/056.000; 935/058.000; 935/061.000; 935/073.000
NCL NCLM: 435/006.000
NCLS: 435/007.320; 435/007.340; 435/034.000; 435/036.000; 435/069.100; 435/235.100; 435/252.300; 435/252.330; 435/320.100; 435/488.000; 530/350.000; 536/023.530; 536/024.100
IC [5]
ICM: C12Q001-68
ICS: C12Q001-00; C12P021-02; C12P019-34; C12N015-00; C12N007-00; C12N001-21; C12N015-70; C12N015-72; C07H015-12; C07K003-00
EXF 435/68; 435/172.1; 435/172.3; 435/252.33; 435/320.1; 435/69.1; 435/235.1; 435/91; 435/7.1; 435/7.2; 435/235.1; 536/27; 530/350; 935/19; 935/31; 935/41; 935/58; 935/60; 935/73; 935/81
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 32 OF 33 USPATFULL
AN 91:38413 USPATFULL
TI DNA encoding hyaluronate synthase
IN Weigel, Paul H., Dickinson, TX, United States

Papaconstantinou, John, Galveston, TX, United States
PA Board of Regents, The University of Texas System, Austin, TX, United States (U.S. corporation)
PI US 5015577 19910514
AI US 1989-401316 19890829 (7)
DT Utility
FS Granted
LN.CNT 1321
INCL INCLM: 435/101.000
INCLS: 435/193.000; 435/252.300; 435/252.330; 435/320.100; 435/849.000; 435/885.000; 536/026.000; 536/027.000; 536/028.000; 935/022.000; 935/027.000; 935/055.000; 935/056.000; 935/060.000; 935/072.000; 935/073.000
NCL NCLM: 435/101.000
NCLS: 435/193.000; 435/252.300; 435/252.330; 435/320.100; 435/849.000; 435/885.000; 536/023.200; 536/023.700
IC [5]
ICM: C12P019-04
ICS: C12N001-20; C12N009-10; C07H015-12
EXF 435/101; 435/104; 435/252.3; 435/252.33; 435/320; 435/849; 435/885; 435/172.3; 536/123; 536/28
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L5 ANSWER 33 OF 33 USPATFULL
AN 86:73220 USPATFULL
TI Transposon in cloning DNA
IN Clewell, Don B., Ann Arbor, MI, United States
Gawron-Burke, Mary C., Ann Arbor, MI, United States
PA Board of Regents of The University of Michigan, Ann Arbor, MI, United States (U.S. corporation)
PI US 4631259 19861223
AI US 1983-491352 19830504 (6)
DT Utility
FS Granted
LN.CNT 586
INCL INCLM: 435/172.300
INCLS: 435/068.000; 435/317.000; 935/023.000; 935/038.000; 935/056.000; 935/073.000
NCL NCLM: 435/473.000
NCLS: 435/069.200; 435/069.300; 435/069.400; 435/069.500; 435/069.510; 435/069.520; 435/069.600; 435/069.700; 435/320.100
IC [4]
ICM: C12P021-00
ICS: C12N001-00; C12N015-00
EXF 435/172.2; 435/172.3; 435/253; 435/832; 435/848; 435/885; 435/886
CAS INDEXING IS AVAILABLE FOR THIS PATENT

9/494297

```
Trying 3106016892...Open

Welcome to STN International!  Enter x:x
LOGINID:ssspta1813nxm
PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2


* * * * * * * * * *   Welcome to STN International   * * * * * * * * * *

 NEWS  1           Web Page URLs for STN Seminar Schedule - N. America
 NEWS  2  Dec 17   The CA Lexicon available in the CAPLUS and CA files
 NEWS  3  Feb 06   Engineering Information Encompass files have new names
 NEWS  4  Feb 16   TOXLINE no longer being updated
 NEWS  5  Apr 23   Search Derwent WPINDEX by chemical structure
 NEWS  6  Apr 23   PRE-1967 REFERENCES NOW SEARCHABLE IN CAPLUS AND CA
 NEWS  7  May 07   DGENE Reload
 NEWS  8  Jun 20   Published patent applications (A1) are now in USPATFULL

 NEWS EXPRESS  May 23 CURRENT WINDOWS VERSION IS V6.0a,
               CURRENT MACINTOSH VERSION IS V5.0C (ENG) AND V5.0JB (JP),
               AND CURRENT DISCOVER FILE IS DATED 06 APRIL 2001
 NEWS HOURS    STN Operating Hours Plus Help Desk Availability
 NEWS INTER    General Internet Information
 NEWS LOGIN    Welcome Banner and News Items
 NEWS PHONE    Direct Dial and Telecommunication Network Access to STN
 NEWS WWW      CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on that
specific topic.

  All use of STN is subject to the provisions of the STN Customer
  agreement.  Please note that this agreement limits use to scientific
  research.  Use for software development or design or implementation
  of commercial gateways or other similar uses is prohibited and may
  result in loss of user privileges and other penalties.

  * * * * * * * * * * * * * * STN  Columbus  * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 15:44:35 ON 21 JUN 2001

=> file uspatfull wpids caplus agricola

COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     0.15       0.15

FILE 'USPATFULL' ENTERED AT 15:45:05 ON 21 JUN 2001
CA INDEXING COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'WPIDS' ENTERED AT 15:45:05 ON 21 JUN 2001
COPYRIGHT (C) 2001 DERWENT INFORMATION LTD

FILE 'CAPLUS' ENTERED AT 15:45:05 ON 21 JUN 2001
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2001 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'AGRICOLA' ENTERED AT 15:45:05 ON 21 JUN 2001
```

```
=> e podbielski andreas/au

E1            1     PODBIELSKA ZOFIA/AU
E2           26     PODBIELSKI A/AU
E3           53 --> PODBIELSKI ANDREAS/AU
E4            4     PODBIELSKI ANGELA/AU
E5            1     PODBIELSKI BASIA/AU
E6            1     PODBIELSKI E/AU
E7            1     PODBIELSKI E P/AU
E8            1     PODBIELSKI EUGENE P/AU
E9            1     PODBIELSKI F J/AU
E10           1     PODBIELSKI FRANCIS J/AU
E11           2     PODBIELSKI GISELE/AU
E12           2     PODBIELSKI J/AU

=> s e2-e4

L1           83 ("PODBIELSKI A"/AU OR "PODBIELSKI ANDREAS"/AU OR "PODBIELSKI
                ANGELA"/AU)

=> s l1 and collagen

L2            1 L1 AND COLLAGEN

=> d bib ab


L2   ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2001 ACS
AN   1999:159061  CAPLUS
DN   131:14761
TI   Characterization of nra, a global negative regulator gene in group A
     streptococci
AU   Podbielski, Andreas; Woischnik, Markus; Leonard, Bettina A. B.;
     Schmidt, Karl-Hermann
CS   Department of Medical Microbiology and Hygiene, University Hospital Ulm,
     Ulm, D-89081, Germany
SO   Mol. Microbiol. (1999), 31(4), 1051-1064
     CODEN: MOMIEE; ISSN: 0950-382X
PB   Blackwell Science Ltd.
DT   Journal
LA   English
AB   During sequencing of an 11.5 kb genomic region of a serotype M49 group A
     streptococcal (GAS) strain, a series of genes were identified including
     nra (neg. regulator of GAS).  Transcriptional anal. of the region
revealed
     that nra was primarily monocistronically transcribed.  Polycistronic
     expression was found for the three open reading frames (ORFs) downstream
     and for the four ORFs upstream of nra.  The deduced Nra protein sequence
     exhibited 62% homol. to the GAS RofA pos. regulator.  In contrast to
RofA,
     Nra was found to be a neg. regulator of its own expression and that of
the
     two adjacent operons by anal. of insertional inactivation mutants.  By
     polymerase chain reaction and hybridization assays of 10 different GAS
     serotypes, the genomic presence of nra, rofA or both was demonstrated.
     Nra-regulated genes include the fibronectin-binding protein F2 gene
     (prtF2) and a novel collagen-binding protein (cpa).  The Cpa
     polypeptide was purified as a recombinant maltose-binding protein fusion
     and shown to bind type I collagen but not fibronectin.  In
     accordance with nra acting as a neg. regulator of prtF2 and cpa, levels
of
     attachment of the nra mutant strain to immobilized collagen and
     fibronectin was increased above wild-type levels.  In addn., nra was also
     found to regulate neg. (four- to 16-fold) the global pos. regulator gene,
```

mga. Using a strain carrying a chromosomally integrated duplication of the nra 3' end and an nra-luciferase reporter gene transcriptional fusion,
nra expression was obsd. to reach its max. during late logarithmic growth phase, while no significant influence of atm. conditions could be distinguished clearly.
RE.CNT 53
RE
(2) Brakhage, A; Biochimie 1990, V72, P725 CAPLUS
(3) Caparon, M; J Bacteriol 1992, V174, P5693 CAPLUS
(4) Caparon, M; Methods Enzymol 1991, V204, P556 CAPLUS
(5) Chen, C; Mol Gen Genet 1993, V241, P685 CAPLUS
(6) Chen, D; J Biol Chem 1994, V269, P32120 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s l1 and streptococc?

L3 60 L1 AND STREPTOCOCC?

=> s l3 and binding (5a) protein

L4 17 L3 AND BINDING (5A) PROTEIN

=> dup rem l4

PROCESSING COMPLETED FOR L4
L5 17 DUP REM L4 (0 DUPLICATES REMOVED)

=> d bib ab 1-17

L5 ANSWER 1 OF 17 CAPLUS COPYRIGHT 2001 ACS
AN 2001:20287 CAPLUS
DN 134:219619
TI Group A **streptococcal** rofA gene is involved in the control of several virulence genes and eukaryotic cell attachment and internalization
AU Beckert, Susanne; Kreikemeyer, Bernd; **Podbielski, Andreas**
CS Department of Medical Microbiology, University Hospital Ulm, Ulm, D-89081,
Germany
SO Infect. Immun. (2001), 69(1), 534-537
CODEN: INFIBR; ISSN: 0019-9567
PB American Society for Microbiology
DT Journal
LA English
AB The serotype M6 group A **streptococcal** RofA regulator was previously shown to exert a direct pos. control of **protein** F1 expression and, concomitantly, fibronectin **binding**. Using a serotype M6 rofA mutant, we demonstrate here that this regulator has a potentially indirect neg. influence on the expression of the mga, emm6, pel-sagA, and speA virulence genes. Addnl., the rofA mutant exhibited reduced eukaryotic cell internalization rates in combination with decreased host cell viability.
RE.CNT 35
RE
(1) Courtney, H; Infect Immun 1994, V62, P3937 CAPLUS
(2) Fogg, G; J Bacteriol 1997, V179, P6172 CAPLUS
(3) Fogg, G; Mol Microbiol 1994, V11, P671 CAPLUS
(4) Gibson, C; J Bacteriol 1996, V178, P4688 CAPLUS
(5) Goodfellow, A; J Clin Microbiol 2000, V38, P389 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 2 OF 17 CAPLUS COPYRIGHT 2001 ACS

AN 2001:104920 CAPLUS
TI Group A **streptococcal** growth phase-associated virulence factor regulation by a novel operon (Fas) with homologies to two-component-type regulators requires a small RNA molecule
AU Kreikemeyer, Bernd; Boyle, Michael D. P.; Buttaro, Bettina A.; Heinemann, Markus; **Podbielski, Andreas**
CS Department of Medical Microbiology and Hygiene, University Hospital Ulm, Ulm, D-89081, Germany
SO Mol. Microbiol. (2001), 39(2), 392-406
CODEN: MOMIEE; ISSN: 0950-382X
PB Blackwell Science Ltd.
DT Journal
LA English
AB A novel growth phase-assocd. two-component-type regulator, Fas (fibronectin/fibrinogen binding/haemolytic activity/streptokinase regulator), of **Streptococcus** pyogenes was identified in the M1 genome sequence, based on homologies to the histidine protein kinase (HPK) and response regulator (RR) part of the Staphylococcus aureus Agr and **Streptococcus** pneumoniae Com quorum-sensing systems. The fas operon, present in all 12 tested M serotypes, was transcribed as polycystronic message (fasBCA) and contained genes encoding two potential HPKs (FasB and FasC) and one RR (FasA). Downstream of fasBCA, we identified a small 300 nucleotide monocistronic transcript, designated fasX, that did not appear to encode true peptide sequences. Measurements of luciferase promoter fusions revealed a growth phase-assocd. transcription of fasBCA and fasX, with peak activities during the late exponential phase. Insertional mutagenesis disrupting fasBCA and fasA led to a phenotype similar to agr-null mutations in S. aureus, with prolonged expression of extracellular matrix **protein-binding** adhesins and reduced expression of secreted virulence factors such as streptokinase and streptolysin S. In addn., fasX transcription was dependent on the RR FasA; however, deletion mutagenesis of fasX resulted in a similar phenotype to that of the fasBCA or fasA mutants. Complementation of the fasX deletion mutant, with the fasX gene expressed in trans from a plasmid, restored the wild-type fasBCA regulation pattern. This strongly suggested that fasX, a putative non-translated RNA, is the main effector mol. of the fas regulon. However, using spent culture supernatants from wild-type and fas mutant strains, we were not able to show an influence on the logarithmic growth phase expression of fas and dependent genes. Thus, despite structural and functional similarities between fas and agr, to date the fas operon appears not to be involved in group A **streptococcal** (GAS) quorum-sensing regulation.
RE.CNT 63
RE
(1) Aarons, S; J Bacteriol 2000, V182, P3913 CAPLUS
(4) Baev, D; Infect Immun 1999, V67, P4510 CAPLUS
(5) Bernish, B; J Biol Chem 1999, V274, P4786 CAPLUS
(6) Betschel, S; Infect Immun 1998, V66, P1671 CAPLUS
(7) Breton, R; J Biol Chem 1990, V265, P18248 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 3 OF 17 CAPLUS COPYRIGHT 2001 ACS
AN 1999:234003 CAPLUS
DN 130:277670
TI Nucleic acid mol. encoding **Streptococcus** agalactiae gene lmb Lmb adhesion mediator, its DNA sequence, and use in prodn. of recombinant of Lmb polypeptide
IN Spellerberg, Barbara; Lutticken, Rudolf; **Podbielski, Andreas**; Rozdzinski, Eva
PA Medimmune, Inc., USA
SO PCT Int. Appl., 69 pp.
CODEN: PIXXD2

DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9916882	A1	19990408	WO 1998-US20028	19980925
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	AU 9895076	A1	19990423	AU 1998-95076	19980925
	EP 1037997	A1	20000927	EP 1998-948522	19980925
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, NL, SE, PT, IE, FI				
PRAI	US 1997-59952	P	19970926		
	WO 1998-US20028	W	19980925		

AB The present invention provides nucleic acid mols. isolated from **Streptococcus** agalactiae encoding gene lmb (laminin binding) polypeptides, identified as Lmb **streptococcal** adhesion mediators. The DNA sequence of S. agalactiae gene lmb is claimed, as well as the amino acid sequence of the Lmb polypeptide. The invention also provided a process for producing a recombinant vector contg. polynucleotides encoding gene lmb, and use of vector in the recombinant expression of the Lmb polypeptides. Further, the invention provides for a vaccine composed of either an immunogenic portion of S. agalactiae Lmb polypeptide or a host cell or vector capable of expressing the immunogenic portion of Lmb in vivo. The DNA was cloned using a genomic library obtained from Group B **Streptococcus** R268 and found to encode a mature protein of 290 amino acids. Transcription anal. of the lmb gene demonstrated that lmb is part of an operon. Potential uses of the Lmb polynucleotides and polypeptides were discussed in the invention.

RE.CNT 3
RE
(1) Jenkinson, H; FEMS MICROBIOL LETTERS 1994, V121(2), P133 CAPLUS
(2) Podbielski, A; DATABASE GENBANK Accession No X80397
(3) Spellerberg, B; Abstracts of the 97th General Meeting of the American Society for Microbiology 1997, P36

L5 ANSWER 4 OF 17 CAPLUS COPYRIGHT 2001 ACS
AN 1999:159061 CAPLUS
DN 131:14761
TI Characterization of nra, a global negative regulator gene in group A **streptococci**
AU **Podbielski, Andreas**; Woischnik, Markus; Leonard, Bettina A. B.; Schmidt, Karl-Hermann
CS Department of Medical Microbiology and Hygiene, University Hospital Ulm, Ulm, D-89081, Germany
SO Mol. Microbiol. (1999), 31(4), 1051-1064
CODEN: MOMIEE; ISSN: 0950-382X
PB Blackwell Science Ltd.
DT Journal
LA English
AB During sequencing of an 11.5 kb genomic region of a serotype M49 group A **streptococcal** (GAS) strain, a series of genes were identified including nra (neg. regulator of GAS). Transcriptional anal. of the region revealed that nra was primarily monocistronically transcribed. Polycistronic expression was found for the three open reading frames (ORFs) downstream and for the four ORFs upstream of nra. The deduced Nra protein sequence exhibited 62% homol. to the GAS RofA pos. regulator. In

contrast to RofA, Nra was found to be a neg. regulator of its own expression and that of the two adjacent operons by anal. of insertional inactivation mutants. By polymerase chain reaction and hybridization assays of 10 different GAS serotypes, the genomic presence of nra, rofA or both was demonstrated. Nra-regulated genes include the fibronectin-**binding protein** F2 gene (prtF2) and a novel collagen-**binding protein** (cpa). The Cpa polypeptide was purified as a recombinant maltose-**binding protein** fusion and shown to bind type I collagen but not fibronectin. In accordance with nra acting as a neg. regulator of prtF2 and cpa, levels of attachment of the nra mutant strain to immobilized collagen and fibronectin was increased above wild-type levels. In addn., nra was also found to regulate neg. (four- to 16-fold) the global pos. regulator gene, mga. Using a strain carrying a chromosomally integrated duplication of the nra 3' end and an nra-luciferase reporter gene transcriptional fusion, nra expression was obsd. to reach its max. during late logarithmic growth phase, while no significant influence of atm. conditions could be distinguished clearly.

RE.CNT 53

RE

(2) Brakhage, A; Biochimie 1990, V72, P725 CAPLUS
(3) Caparon, M; J Bacteriol 1992, V174, P5693 CAPLUS
(4) Caparon, M; Methods Enzymol 1991, V204, P556 CAPLUS
(5) Chen, C; Mol Gen Genet 1993, V241, P685 CAPLUS
(6) Chen, D; J Biol Chem 1994, V269, P32120 CAPLUS

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 5 OF 17 CAPLUS COPYRIGHT 2001 ACS
AN 1998:617772 CAPLUS
DN 129:328090
TI An ectoprotein kinase of group C **streptococci** binds hyaluronan and regulates capsule formation
AU Nickel, Volker; Prehm, Sabine; Lansing, Manfred; Mausolf, Andreas; **Podbielski, Andreas**; Deutscher, Josef; Prehm, Peter
CS Institut fur Physiologische Chemie und Pathobiochemie, Munster, D-48129, Germany
SO J. Biol. Chem. (1998), 273(37), 23668-23673
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
AB A 56-kDa protein had been isolated and cloned from protoplast membranes of group C **streptococci** that had erroneously been identified as hyaluronan synthase. The function of this protein was reexamd. When **streptococcal** membranes were sepd. on an SDS-polyacrylamide gel and renatured, a 56-kDa protein was detected that had kinase activity for a casein substrate. When this recombinant protein was expressed in Escherichia coli and incubated in the presence of [32P]ATP, it was responsible for phosphorylation of two proteins with 30 and 56 kDa that were not present in the control lysate. The 56-kDa protein was specifically phosphorylated in an immunoppt. of a detergent ext. of the recombinant E. coli lysate with antibodies against the 56-kDa protein, indicating that it was autophosphorylated. The E. coli lysate contg. the recombinant **protein** could bind hyaluronan, and hyaluronan **binding** was abolished by the addn. of ATP. Kinetic anal. of hyaluronan synthesis and release from isolated protoplast membranes indicated that phosphorylation by ATP stimulated hyaluronan release and synthesis. Incubation of membranes with antibodies to the 56-kDa protein increased hyaluronan release. The addn. of [32P]ATP to intact **streptococci** led to rapid phosphorylation of two proteins, 56 and 75 kDa each at threonine residues. This phosphorylation was neither obsd. with [32P]phosphate nor in the presence of trypsin, indicating that the

kinase was localized extracellularly. The addn. of ATP to growing group C **streptococci** led to increased hyaluronan synthesis and release. However marked differences were found between group A and group C **streptococci**. Antibodies against the 56-kDa protein from group C **streptococci** did not recognize proteins from group A strains, and a homologous DNA sequence could not be detected by polymerase chain reaction or Southern blotting. In addn., Group A **streptococci** did not retain a large hyaluronan capsule like group C strains. These results indicated that the 56-kDa protein is an ectoprotein kinase specific for group C **streptococci** that regulates hyaluronan capsule shedding by phosphorylation.

L5 ANSWER 6 OF 17 CAPLUS COPYRIGHT 2001 ACS
AN 1998:708685 CAPLUS
DN 130:107378
TI A secreted **streptococcal** cysteine protease can cleave a surface-expressed M1 **protein** and alter the immunoglobulin **binding** properties
AU Raeder, R.; Woischnik, M.; **Podbielski, A.**; Boyle, M. D. P.
CS Department of Microbiology and Immunology, Medical College of Ohio, Toledo, OH, 43699-0008, USA
SO Res. Microbiol. (1998), 149(8), 539-548
CODEN: RMCREW; ISSN: 0923-2508
PB Editions Scientifiques et Medicales Elsevier
DT Journal
LA English
AB Previous studies of recent clin. isolates of serotype M1 group A **streptococci** indicated that they display 2 patterns of non-immune human IgG subclass **binding** reactivity assocd. with their M1 **protein**. One group reacted with all 4 IgG subclasses (type IIo), while the 2nd group expressed an M1 protein reacting preferentially with human IgG3 (type IIb). This study demonstrates that a cysteine protease, SpeB, present in culture supernatants of M1 serotype group A **streptococcal** isolates expressing type IIb IgG **binding** **protein**, can convert a recombinant Emm1 protein from a type IIo functional profile to a type IIb profile by removal of 24 amino acids from the N-terminus of the mature M1 protein. Furthermore, SpeB can convert bacteria expressing IgG-binding proteins of the type IIo phenotype into those expressing type IIb proteins. The role of the cysteine protease as the central bacterial enzyme in this post-translational modification event was confirmed by generation of an isogenic SpeB-neg. mutant.
RE.CNT 28
RE
(1) Barrett, A; Biochem J 1982, V201, P189 CAPLUS
(2) Bayer, E; Meth Enzymol 1990, V184, P138 CAPLUS
(3) Berge, A; J Biol Chem 1995, V270, P9862 CAPLUS
(4) Burns, E; Infect Immun 1996, V64, P4744 CAPLUS
(5) Chaussee, M; Infect Immun 1993, V61, P3719 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 7 OF 17 CAPLUS COPYRIGHT 2001 ACS
AN 1997:306383 CAPLUS
DN 126:340850
TI Identification of key gene products required for acquisition of plasmin-like enzymic activity by group A **streptococci**
AU Christner, Robert; Li, Zhuqing; Raeder, Roberta; **Podbielski, Andreas**; Boyle, Michael D. P.
CS Department of Microbiology, Medical College of Ohio, Toledo, OH, 43699-0008, USA
SO J. Infect. Dis. (1997), 175(5), 1115-1120
CODEN: JIDIAQ; ISSN: 0022-1899
PB University of Chicago Press

DT Journal
LA English
AB Group A **streptococci** incubated in human plasma can acquire a plasmin-like enzymic activity. This process involves at least two bacterial proteins and two human protein cofactors. In this study, the key bacterial proteins were identified by using a series of isogenic mutants of group A isolate, CS101. These studies confirm a key role for the secreted plasminogen activator, streptokinase, and identify the major surface fibrinogen-**binding protein** as the product of the mrp gene. The requirement for human fibrinogen and plasminogen as key cofactors was also confirmed.

L5 ANSWER 8 OF 17 CAPLUS COPYRIGHT 2001 ACS
AN 1998:128759 CAPLUS
DN 128:228323
TI Inactivation of single genes within the virulence regulon of an M2 group A **streptococcal** isolate results in differences in virulence for chicken embryos and for mice
AU Schmidt, Karl-Hermann; **Podbielski, Andreas**; Raeder, Roberta; Boyle, Michael D. P.
CS Institute of Medical Microbiology, Hospital of Jena, Jena, D-07740, Germany
SO Microb. Pathog. (1997), 23(6), 347-355
CODEN: MIPAEV; ISSN: 0882-4010
PB Academic Press Ltd.
DT Journal
LA English
AB An M2 **streptococcal** isolate and isogenic mutants in which either the emm or mrp gene was insertionally inactivated were tested for virulence using either a mouse model or a chicken embryo model. The results of the studies using the mouse model demonstrated that neither the emm nor mrp gene products had a significant effect on virulence when mice were challenged via the i.p. route. However, when the bacteria were injected into the skin the emm gene product was identified as a virulence factor. In parallel studies in the chicken embryo model the mrp gene product was found to be a major virulence factor, while a minor contribution to virulence could also be attributed to the emm gene product. The importance of these gene products to virulence was noted when the chicken embryo were injected either i.v or when the bacteria were placed on top of the chorioallantoic membrane. The direct comparison of a single wild type group A organism and its paired isogenic mutants in two animal models suggests that different combinations of bacterial factors are required to overcome host defense strategies assocd. with different animal species.

L5 ANSWER 9 OF 17 CAPLUS COPYRIGHT 2001 ACS
AN 1996:563892 CAPLUS
DN 125:239725
TI Molecular characterization of group A **streptococcal** (GAS) oligopeptide permease (Opp) and its effect on cysteine protease production
AU **Podbielski, Andreas**; Pohl, Barbara; Woischnik, Markus; Koerner, Christiane; Schmidt, Karl-Hermann; Rozdzinski, Eva; Leonard, Bettina A. B.
CS Inst. Med. Microbiology, Hospital Technical Univ., Aachen, D-52057, Germany
SO Mol. Microbiol. (1996), 21(5), 1087-1099
CODEN: MOMIEE; ISSN: 0950-382X
DT Journal
LA English

AB Bacterial oligopeptide permeases are membrane-assocd. complexes of five proteins belonging to the ABC-transporter family, which have been found to be involved in obtaining nutrients, cell-wall metab., competence, and adherence to host cells. A lambda library of the strain CS101 group A **streptococcal** (GAS) genome was used to sequence 10 192 bp contg. the five genes oppA to oppF of the GAS opp operon. The deduced amino acid sequences exhibited 50-84% homol. to pneumococcal AmiA to AmiF sequences. The operon organization of the five genes was confirmed by transcriptional anal. and an addnl. shorter oppA transcript was detected. Insertional inactivation was used to create serotype M49 strains which did not express either the oppA gene or the ATPase genes, oppD and oppF. The mutation in oppA confirmed that the addnl. shorter oppA transcript originated from the opp operon and was probably due to an intra-operon transcription terminator site located downstream of oppA. While growth kinetics, binding of serum proteins, and attachment to eukaryotic cells were unaffected, the oppD/F mutants showed reduced prodn. of the cysteine protease, SpeB, and a change in the pattern of secreted proteins. Thus, the GAS opp operon appears to contribute to both protease prodn. and export/processing of secreted proteins.

L5 ANSWER 10 OF 17 CAPLUS COPYRIGHT 2001 ACS
AN 1996:347844 CAPLUS
DN 125:55964
TI Identification of an amino acid signature sequence predictive of **protein** G-inhibitable IgG3-**binding** activity in group-A **streptococcal** IgG-binding proteins
AU Pack, Todd D.; **Podbielski, Andreas**; Boyle, Michael D. P.
CS Department of Microbiology, Medical College of Ohio, Toledo, OH, 43699-0008, USA
SO Gene (1996), 171(1), 65-70
CODEN: GENED6; ISSN: 0378-1119
DT Journal
LA English
AB Sequence comparison of six known group-A **streptococcal** IgG-binding proteins, sharing the common property of **protein** G-inhibitable IgG3-**binding**-activity, identified a highly conserved 35-amino-acid (aa) sequence (74-100% similarity) within an EQ-rich central conserved core region of each protein. A search of aa sequence databases identified four addnl. proteins with >50% similarity to this consensus sequence. All of these proteins demonstrated **protein** G-inhibitable IgG3-**binding** activity. Taken together, these results identify a signature sequence that predicts the presence of a **protein** G-inhibitable IgG3-**binding** domain(s) in group-A **streptococcal** IgG-binding proteins.

L5 ANSWER 11 OF 17 CAPLUS COPYRIGHT 2001 ACS
AN 1995:722009 CAPLUS
DN 123:280393
TI Characterization of a gene coding for a type IIo bacterial IgG-**binding protein**
AU Boyle, Michael D. P.; Weber-Heynemann, Josephine; Raeder, Roberta; **Podbielski, Andreas**
CS Department Microbiology, Medical College of Ohio, Toledo, OH, 43699-0008, USA
SO Mol. Immunol. (1995), 32(9), 669-78
CODEN: MOIMD5; ISSN: 0161-5890
DT Journal
LA English
AB Two antigenic classes of non-immune IgG-binding proteins can be expressed

by group A **streptococci**. One antigenic group of proteins is recognized by an antibody prepd. against the product of a cloned fcrA gene (anti-FcRA). In this study, the immunogen used to prep. the antibody that defines the second antigenic class was shown to be the product of the emm-like (emmL) gene of M serotype 55 group A isolate, A928. The emmL55 gene expressed in E. coli produced an Mr .apprx. 58,000 mol. which bound human IgG1, IgG2, IgG3 and IgG4, as well as horse, rabbit and pig IgG in a non-immune fashion. These properties are characteristic of the previously described type IIo IgG-**binding protein** isolated from this strain. In addn., the recombinant protein was reactive with human serum albumin and fibrinogen. The emmL 55 gene sequence was analyzed and found to have the organization and sequence characteristics of a typical class I emm-like gene.

L5 ANSWER 12 OF 17 CAPLUS COPYRIGHT 2001 ACS
AN 1995:4550 CAPLUS
DN 122:3780
TI Analysis of genes encoding two unique type IIa immunoglobulin G-binding proteins expressed by a single group A **streptococcal** isolate
AU Boyle, Michael D. P.; Hawlitzky, Joerg; Raeder, Roberta; **Podbielski, Andreas**
CS Dep. Microbiol., Med. Coll. Ohio, Toledo, OH, 43699-0008, USA
SO Infect. Immun. (1994), 62(4), 1336-47
CODEN: INFIBR; ISSN: 0019-9567
DT Journal
LA English
AB An emm-like gene (emmL) and a fcrA gene from group A **streptococcal** strain 64/14 (emmL64/14 and fcrA64/14) were amplified by PCR and force cloned into the heat-inducible expression vector pJLA 602. The emmL gene encoded a recombinant protein that bound human IgG1, IgG2, and IgG4 in a nonimmune fashion. This is the reactivity profile of a type IIa IgG-**binding protein**. The emmL64/14 gene product was antigenically similar to the previously identified high-mol.-wt. type IIa IgG-**binding protein** of strain 64/14 and had an N-terminal sequence identical to that of the wild-type protein. The fcrA gene also encoded a recombinant protein with type IIa functional activity. This protein was similar to the lower-mol.-wt. type IIa IgG-**binding protein** previously isolated from strain 64/14 and was antigenically distinct from the higher-mol.-wt. type IIa protein encoded by the emmL64/14 gene. The sequences for both genes including the intervening regions are presented. The emmL gene demonstrates significant homol. to other class I emm and emmL gene expressed by opacity factor-neg. group A **streptococcal** isolates. The fcrA gene was found to be homologous to other fcrA genes normally present in opacity factor-pos. group A isolates. The sequence upstream of the fcrA gene and the intervening sequence between the end of the fcrA gene and the start of the emmL gene were similar to those reported for other fcrA genes.

L5 ANSWER 13 OF 17 CAPLUS COPYRIGHT 2001 ACS
AN 1994:550413 CAPLUS
DN 121:150413
TI Genetic variability of the emm-related genes of the large vir regulon of group A **streptococci**: potential intra- and intergenomic recombination events
AU **Podbielski, A.**; Krebs, B.; Kaufhold, A.
CS Inst. Medical Microbiology, Technical Univ., Aachen, D-52057, Germany

SO Mol. Gen. Genet. (1994), 243(6), 691-698
CODEN: MGGEAE; ISSN: 0026-8925
DT Journal
LA English
AB One of the most prevalent genetic lineages of group A **streptococci** (GAS) harbors a genomic locus termed the large vir regulon, which contains an emm gene encoding the antiphagocytic M protein, and structurally related fcrA and enn (emm-related) genes encoding Ig-binding proteins. In the present study more than 100 large vir regulons from 42 different GAS serotypes were analyzed by PCR and partial DNA sequencing. On comparing these data to published sequences, sites of mutational and putative recombinational events were identified and ordered with respect to their intra/intergenic or intra/intergenomic nature. The emm-related genes were found to display small intragenic deletions or insertions, were completely deleted from, or newly inserted into the genome, or were fused to adjacent genes. Intergenomic exchanges of complete emm-related genes, or segments thereof, between different vir regulons were detected. Most of these processes seem to involve short flanking direct repeats. Occasionally, the structural changes could be correlated with changes in the functions of the encoded proteins.

L5 ANSWER 14 OF 17 CAPLUS COPYRIGHT 2001 ACS
AN 1995:10590 CAPLUS
DN 122:3792
TI Immunoglobulin-binding FcrA and Enn proteins and M proteins of group A **streptococci** evolved independently from a common ancestral protein
AU **Podbielski, Andreas**; Weber-Heynemann, Josephine; Cleary, Patrick P.
CS Dep. Med. Microbiol., Tech. Univ., Aachen, D-52057, Germany
SO Med. Microbiol. Immunol. (1994), 183(1), 33-42
CODEN: MMIYAO; ISSN: 0300-8584
DT Journal
LA English
AB Significant sequence homol. between M proteins and Ig-binding proteins of group A **streptococci** suggests that these proteins arose by gene duplication followed by the development of functional diversity due to mutations and intragenic recombinations. The deduced sequence of multiple Ig-binding proteins and M proteins were compared to distinguish between 2 evolutionary models. Did these functionally distinct genes originate in the distant past from duplication of a common ancestral gene and then functionally evolve independently or did they evolve more recently, one from the other by duplication of a fixed gene. Multiple alignments of conserved sequences of these proteins are consistent with the former hypothesis. Comparison of N termini of Ig-binding proteins revealed less diversity than that of the M proteins' N termini, suggesting that these proteins are under less selective pressure to change.

L5 ANSWER 15 OF 17 CAPLUS COPYRIGHT 2001 ACS
AN 1993:231908 CAPLUS
DN 118:231908
TI Type M12 protein from **Streptococcus** pyogenes is a receptor for IgG3
AU Retnoningrum, Debbie S.; **Podbielski, Andreas**; Cleary, P. Patrick
CS Dep. Microbiol., Univ. Minnesota, Minneapolis, MN, 55455, USA
SO J. Immunol. (1993), 150(6), 2332-40
CODEN: JOIMA3; ISSN: 0022-1767
DT Journal
LA English
AB Serotype M12 S. pyogenes was discovered to express a human IgG3

binding protein. Western blot anal. of partially purified M12 protein, exposed to IgG3 myeloma protein, showed that both M12 antigen (Ag) and the receptor protein were the same apparent size. A .lambda. clone (.lambda.4.1) contg. the emm12 open reading frame expressed both the M12 Ag and the IgG3 **binding protein**. The emm12 open reading frame was amplified by the polymerase chain reaction and subcloned into the expression vector pJLA602. Based on Western blot anal., 1 recombinant Escherichia coli (pD3) expressed M12 **protein** with IgG3 **binding** activity. This result confirmed that the M12 protein from strain CS24 is also an IgG3 receptor. Deletion analyses showed that a truncated M12 protein encoded by an internal PvuII fragment was sufficient for IgG3 binding activity. Further deletion studies suggested that the IgG3 binding domain was located in a 200-amino acid internal fragment contg. 2 directly repeated sequences. Other expts. suggest that the receptor did not bind to the same IgG3 domain as that recognized by protein G. The M12 protein did not bind human IgG1, IgG2, IgG4, or Ig from several other animal species.

L5 ANSWER 16 OF 17 CAPLUS COPYRIGHT 2001 ACS
AN 1993:464499 CAPLUS
DN 119:64499
TI Three different types of organization of the vir regulon in group A **streptococci**
AU **Podbielski, Andreas**
CS Inst. Med. Microbiol., Tech. Univ., Aachen, W-5100, Germany
SO Mol. Gen. Genet. (1993), 237(1-2), 287-300
CODEN: MGGEAE; ISSN: 0026-8925
DT Journal
LA English
AB The DNA of group A **streptococci** (GAS) encodes several important virulence factors such as the antiphagocytic M **protein**, the Ig-Fc-**binding** M-related proteins (FcrA-like and EnnX-like) and the complement factor-inactivating C5a peptidase. The corresponding genes emm, fcrA, ennX, and scpA, resp., were assumed to be located close together in the GAS genome. Addnl., emm and scpA have been found to be under the pos., coordinate control of the virR locus, which led to the designation "vir regulon" for the corresponding genomic segment. An approach using several distinct sets of PCR expts. was used to map the vir regulons of many GAS serotypes and to analyze any correlation between the organization of vir regulons and circumscribed heterogeneities within the emm, virR, and scpA genes. By examn. of the genomic DNA of 42 GAS isolates from 36 different M serotypes, three patterns of vir regulon topog. were found. The first, designated "large vir regulon" (LVR), consists of virR = fcrA(-like)-emm-ennX(-like)-scpA. The second, designated "small vir regulon" (SVR), contains virR-emm-scpA, and the last, designated "unusual vir regulon" (UVR), resembles SVR but contains addnl. heterogeneous sequences between emm and scpA. The patterns correlate with heterogeneities at the 3' ends of the virR and scpA genes, with the M classification system and the occurrence of specific non-coding intervening sequences within the vir regulons. The potential impact of these patterns on models to account for generation of vir regulations is discussed.

L5 ANSWER 17 OF 17 CAPLUS COPYRIGHT 2001 ACS
AN 1994:403429 CAPLUS
DN 121:3429
TI PCR-mediated amplification of group A **Streptococcal** genes encoding immunoglobulin-binding proteins
AU **Podbielski, A.**; Kaufhold, A.; Cleary, P. P.
CS Inst. Med. Microbiol., Hosp. Tech. Univ., Aachen, 5100, Germany
SO ImmunoMethods (1993), 2(1), 55-64

CODEN: IMUME8; ISSN: 1058-6687
DT Journal
LA English
AB A majority of group A **streptococci** (GAS) express Ig-binding proteins. The genes encoding these proteins belong to either the emm or the emm-related (fcrA and enn) gene family. The present study presents oligonucleotide primers and protocols for the specific PCR-mediated amplification of fcrA and enn genes with and without their own promoters and transcription termination sites. The PCR system appears to be applicable to virtually every GAS serotype. Using these assays, some sequence variability in the 5' untranslated region of fcrA and at the 5' end of enn was demonstrated for a minority of GAS serotypes. The fcrA and enn genes of GAS serotype M49 were amplified by PCR, cloned into pJLA602, and expressed in Escherichia coli. The fcrA gene of the GAS serotype M49 strain CS101, including its adjacent regulatory sequences, was sequenced using a universal primer set by means of which emm-related genes can be completely sequenced with only five sequencing reactions. The fcrA49 sequence provides further evidence that fcrA genes are a sep. entity from emm-related genes and exhibit a variability at their 5' ends similar to that of proteins encoded by emm genes.

=> s streptococc?

L6 55879 STREPTOCOCC?

=> s l6 and collagen

L7 903 L6 AND COLLAGEN

=> s l7 and cpa1

L8 0 L7 AND CPA1

=> s l7 and cpa?

L9 10 L7 AND CPA?

=> dup rem l9

PROCESSING COMPLETED FOR L9
L10 10 DUP REM L9 (0 DUPLICATES REMOVED)

=> d bib ab 1-10

L10 ANSWER 1 OF 10 USPATFULL
AN 2000:95093 USPATFULL
TI Isolated peptides derived from the Epstein-Barr virus containing fusion inhibitory domains
IN Barney, Shawn O'Lin, Cary, NC, United States
Lambert, Dennis Michael, Cary, NC, United States
Petteway, Stephen Robert, Cary, NC, United States
PA Trimeris, Inc., Durham, NC, United States (U.S. corporation)
PI US 6093794 20000725
AI US 1995-471913 19950607 (8)
RLI Division of Ser. No. US 1995-470896, filed on 6 Jun 1995 which is a continuation-in-part of Ser. No. US 1994-360107, filed on 20 Dec 1994 which is a continuation-in-part of Ser. No. US 1994-255208, filed on 7 Jun 1994 which is a continuation-in-part of Ser. No. US 1993-73028, filed on 7 Jun 1993, now patented, Pat. No. US 5464933
DT Utility
EXNAM Primary Examiner: Scheiner, Laurie; Assistant Examiner: Parkin, Jeffrey S.

LREP Pennie & Edmonds LLP
CLMN Number of Claims: 27
ECL Exemplary Claim: 1
DRWN 52 Drawing Figure(s); 83 Drawing Page(s)
LN.CNT 19949
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to peptides which exhibit potent anti-retroviral activity. The peptides of the invention comprise DP178 (SEQ ID:1) peptide corresponding to amino acids 638 to 673 of the HIV-1.sub.LAI gp41 protein, and fragments, analogs and homologs of DP178. The invention further relates to the uses of such peptides as inhibitory of human and non-human retroviral, especially HIV, transmission to uninfected cells.

L10 ANSWER 2 OF 10 USPATFULL
AN 2000:57361 USPATFULL
TI Compositions for inhibition of membrane fusion-associated events, including influenza virus transmission
IN Barney, Shawn O'Lin, Cary, NC, United States
Lambert, Dennis Michael, Cary, NC, United States
Petteway, Stephen Robert, Cary, NC, United States
PA Trimeris, Inc., Durham, NC, United States (U.S. corporation)
Duke University, Durham, NC, United States (U.S. corporation)
PI US 6060065 20000509
AI US 1995-475668 19950607 (8)
RLI Division of Ser. No. US 1995-470896, filed on 6 Jun 1995 which is a continuation-in-part of Ser. No. US 1994-360107, filed on 20 Dec 1994 which is a continuation-in-part of Ser. No. US 1994-255208, filed on 7 Jun 1994 which is a continuation-in-part of Ser. No. US 1993-73028, filed on 7 Jun 1993, now patented, Pat. No. US 5464933
DT Utility
EXNAM Primary Examiner: Achutamurthy, Ponnathapura; Assistant Examiner: Parley, Hankyel T.
LREP Pennie & Edmonds, LLP
CLMN Number of Claims: 5
ECL Exemplary Claim: 1
DRWN 84 Drawing Figure(s); 83 Drawing Page(s)
LN.CNT 19987
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to viral peptides referred to as "DP107- and DP178-like" peptides. Specifically, the invention relates to isolated influenza A DP107- and DP178-like peptides which are identified
by sequence search motif algorithms. The peptides of the invention exhibit antiviral activity believed to result from inhibition of viral induced fusogenic events.

L10 ANSWER 3 OF 10 USPATFULL
AN 2000:50515 USPATFULL
TI Screening assays for compounds that inhibit membrane fusion-associated events
IN Barney, Shawn O'Lin, Cary, NC, United States
Lambert, Dennis Michael, Cary, NC, United States
Petteway, Jr., Stephen Robert, Cary, NC, United States
PA Trimeris, Inc., Durham, NC, United States (U.S. corporation)
PI US 6054265 20000425
AI US 1997-919597 19970926 (8)
RLI Division of Ser. No. US 1995-470896, filed on 6 Jun 1995 which is a continuation-in-part of Ser. No. US 1994-360107, filed on 20 Dec 1994 which is a continuation-in-part of Ser. No. US 1994-255208, filed on 7 Jun 1994 which is a continuation-in-part of Ser. No. US 1993-73028, filed on 7 Jun 1993, now patented, Pat. No. US 5464933
DT Utility
EXNAM Primary Examiner: Stucker, Jeffrey
LREP Pennie & Edmonds, LLP

CLMN Number of Claims: 1
ECL Exemplary Claim: 1
DRWN 83 Drawing Figure(s); 83 Drawing Page(s)
LN.CNT 21307
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to peptides which exhibit potent anti-retroviral activity. The peptides of the invention comprise DP178 (SEQ ID:1) peptide corresponding to amino acids 638 to 673 of the HIV-1.sub.LAI gp41 protein, and fragments, analogs and homologs of DP178. The invention further relates to the uses of such peptides as inhibitory of human and non-human retroviral, especially HIV, transmission to uninfected cells.

L10 ANSWER 4 OF 10 USPATFULL
AN 2000:24479 USPATFULL
TI Fibronectin binding protein as well as its preparation
IN Normark, Staffan, S-913 00 Holmsund, Zackrisvagen, Sweden
Olsen, Arne, 902 41 Umea, Sprakgrand 19, Sweden
PI US 6030805 20000229
AI US 1995-495959 19950628 (8)
RLI Continuation of Ser. No. US 1994-318519, filed on 5 Oct 1994, now abandoned which is a continuation of Ser. No. US 1994-187865, filed on 28 Jan 1994, now abandoned which is a continuation of Ser. No. US 1992-970846, filed on 3 Nov 1992, now abandoned which is a continuation-in-part of Ser. No. US 1991-789437, filed on 6 Nov 1991, now abandoned which is a continuation of Ser. No. US 1989-347189, filed on 4 May 1989, now abandoned
DT Utility
EXNAM Primary Examiner: Wax, Robert A.; Assistant Examiner: Mytelka, Daniel S.
LREP Burns, Doane, Swecker & Mathis, L.L.P.
CLMN Number of Claims: 17
ECL Exemplary Claim: 1
DRWN 7 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 715
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to a new fibronectin binding protein from E. coli in the form of a curli pili. a new recombinant hybrid-DNA-molecule comprising a nucleotide sequence from E. coli coding
for a protein or polypeptide having fibronectin binding properties. (FIG. 4).

L10 ANSWER 5 OF 10 USPATFULL
AN 2000:12922 USPATFULL
TI Isolated peptides derived from human immunodeficiency virus types 1 and 2 containing fusion inhibitory domains
IN Barney, Shawn O'Lin, Cary, NC, United States
Lambert, Dennis Michael, Cary, NC, United States
Petteway, Stephen Robert, Cary, NC, United States
PA Trimeris, Inc., Durham, NC, United States (U.S. corporation)
PI US 6020459 20000201
AI US 1995-484223 19950607 (8)
RLI Division of Ser. No. US 1995-470896, filed on 6 Jun 1995 which is a continuation-in-part of Ser. No. US 1994-360107, filed on 20 Dec 1994 which is a continuation-in-part of Ser. No. US 1994-255208, filed on 7 Jun 1994 which is a continuation-in-part of Ser. No. US 1993-73028, filed on 7 Jun 1993, now patented, Pat. No. US 5464933
DT Utility
EXNAM Primary Examiner: Scheiner, Laurie; Assistant Examiner: Parkin, Jeffrey S.
LREP Pennie & Edmonds LLP
CLMN Number of Claims: 75
ECL Exemplary Claim: 1
DRWN 52 Drawing Figure(s); 83 Drawing Page(s)

LN.CNT 20335
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to peptides which exhibit potent anti-retroviral activity. The peptides of the invention comprise DP178 (SEQ ID:1) peptide corresponding to amino acids 638 to 673 of the HIV-1.sub.LAI gp41 protein, and fragments, analogs and homologs of DP178. The invention further relates to the uses of such peptides as inhibitory of human and non-human retroviral, especially HIV, transmission to uninfected cells.

L10 ANSWER 6 OF 10 USPATFULL
AN 2000:9527 USPATFULL
TI Simian immunodeficiency virus peptides with antifusogenic and antiviral activities
IN Barney, Shawn O'Lin, Cary, NC, United States
Lambert, Dennis Michael, Cary, NC, United States
Petteway, Stephen Robert, Cary, NC, United States
Langlois, Alphonse J., Durham, NC, United States
PA Trimeris, Inc., Durham, NC, United States (U.S. corporation)
PI US 6017536 20000125
AI US 1994-360107 19941220 (8)
RLI Continuation-in-part of Ser. No. US 1994-255208, filed on 7 Jun 1994 which is a continuation-in-part of Ser. No. US 1993-73028, filed on 7 Jun 1993, now patented, Pat. No. US 5464933
DT Utility
EXNAM Primary Examiner: Scheiner, Laurie; Assistant Examiner: Parkin, Jeffrey S.
LREP Pennie & Edmonds LLP
CLMN Number of Claims: 28
ECL Exemplary Claim: 1
DRWN 50 Drawing Figure(s); 62 Drawing Page(s)
LN.CNT 20227
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to peptides which exhibit antifusogenic and antiviral activities. The peptides of the invention consist of a 16 to 39 amino acid region of a simian immunodeficiency virus (SIV) protein. These regions were identified through computer algorithms capable of recognizing the ALLMOTI5, 107.times.178.times.4, or PLZIP amino acid motifs. These motifs are associated with the antifusogenic and antiviral activities of the claimed peptides.

L10 ANSWER 7 OF 10 USPATFULL
AN 2000:4427 USPATFULL
TI Measles virus peptides with antifusogenic and antiviral activities
IN Barney, Shawn O'Lin, Cary, NC, United States
Lambert, Dennis Michael, Cary, NC, United States
Petteway, Stephen Robert, Cary, NC, United States
PA Trimeris, Inc., Durham, NC, United States (U.S. corporation)
PI US 6013263 20000111
AI US 1995-486099 19950607 (8)
RLI Division of Ser. No. US 1995-470896, filed on 6 Jun 1995 which is a continuation-in-part of Ser. No. US 1994-360107, filed on 20 Dec 1994 Ser. No. Ser. No. US 1994-255208, filed on 7 Jun 1994 And Ser. No. US 1993-73028, filed on 7 Jun 1993, now patented, Pat. No. US 5464933
DT Utility
EXNAM Primary Examiner: Scheiner, Laurie; Assistant Examiner: Parkin, Jeffrey S.
LREP Pennie & Edmonds LLP
CLMN Number of Claims: 38
ECL Exemplary Claim: 1
DRWN 52 Drawing Figure(s); 83 Drawing Page(s)
LN.CNT 19827
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to peptides which exhibit potent anti-retroviral activity. The peptides of the invention comprise DP178

(SEQ ID:1) peptide corresponding to amino acids 638 to 673 of the HIV-1.sub.LAI gp41 protein, and fragments, analogs and homologs of DP178. The invention further relates to the uses of such peptides as inhibitory of human and non-human retroviral, especially HIV, transmission to uninfected cells.

L10 ANSWER 8 OF 10 USPATFULL
AN 2000:1692 USPATFULL
TI Sequence-directed DNA binding molecules compositions and methods
IN Edwards, Cynthia A., Menlo Park, CA, United States
Cantor, Charles R., Boston, MA, United States
Andrews, Beth M., Maynard, MA, United States
Turin, Lisa M., Redwood City, CA, United States
Fry, Kirk E., Palo Alto, CA, United States
PA Genelabs Technologies, Inc., Redwood, CA, United States (U.S. corporation)
PI US 6010849 20000104
AI US 1995-482080 19950607 (8)
RLI Division of Ser. No. US 1993-171389, filed on 20 Dec 1993, now patented,
Pat. No. US 5578444 which is a continuation-in-part of Ser. No. US 1993-123936, filed on 17 Sep 1993, now patented, Pat. No. US 5726014 which is a continuation-in-part of Ser. No. US 1992-996783, filed on 23 Dec 1992, now patented, Pat. No. US 5693463 which is a continuation-in-part of Ser. No. US 1991-723618, filed on 27 Jun 1991, now abandoned
DT Utility
EXNAM Primary Examiner: Degen, Nancy; Assistant Examiner: Schwartzman, Robert
LREP Fabin, Gary R.Dehlinger & Associates
CLMN Number of Claims: 11
ECL Exemplary Claim: 1
DRWN 48 Drawing Figure(s); 47 Drawing Page(s)
LN.CNT 10022
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention defines a DNA:protein-binding assay useful for screening libraries of synthetic or biological compounds for their ability to bind DNA test sequences. The assay is versatile in that any number of test sequences can be tested by placing the test sequence adjacent to a defined protein binding screening sequence. Binding of molecules to these test sequence changes the binding characteristics of the protein molecule to its cognate binding sequence. When such a molecule binds the test sequence the equilibrium of the DNA:protein complexes is disturbed, generating changes in the concentration of free DNA probe. Numerous exemplary target test sequences (SEQ ID NO:1 to SEQ ID NO:600) are set forth. The assay of the present invention is also useful to characterize the preferred binding sequences of any selected DNA-binding molecule.

L10 ANSWER 9 OF 10 USPATFULL
AN 1999:18912 USPATFULL
TI Method of determining DNA sequence preference of a DNA-binding molecule
IN Edwards, Cynthia A., Menlo Park, CA, United States
Cantor, Charles R., Boston, MA, United States
Andrews, Beth M., Maynard, MA, United States
Turin, Lisa M., Redwood City, CA, United States
Fry, Kirk E., Palo Alto, CA, United States
PA Genelabs Technologies, Inc., Redwood City, CA, United States (U.S. corporation)
PI US 5869241 19990209
AI US 1995-475228 19950607 (8)
RLI Division of Ser. No. US 1993-171389, filed on 20 Dec 1993, now patented,
Pat. No. US 5578444 which is a continuation-in-part of Ser. No. US 1993-123936, filed on 17 Sep 1993, now patented, Pat. No. US 5726014 which is a continuation-in-part of Ser. No. US 1992-996783, filed on 23

Dec 1992, now patented, Pat. No. US 5693463 which is a continuation-in-part of Ser. No. US 1991-723618, filed on 27 Jun 1991, now abandoned
DT Utility
EXNAM Primary Examiner: Zitomer, Stepanie W.; Assistant Examiner: Whisenant, Ethan
LREP Fabian, Gary R., Stratford, Carol A., Dehlinger, Peter J.
CLMN Number of Claims: 11
ECL Exemplary Claim: 1
DRWN 72 Drawing Figure(s); 47 Drawing Page(s)
LN.CNT 9840
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention defines a DNA:protein-binding assay useful for screening libraries of synthetic or biological compounds for their ability to bind DNA test sequences. The assay is versatile in that any number of test sequences can be tested by placing the test sequence adjacent to a defined protein binding screening sequence. Binding of molecules to these test sequence changes the binding characteristics of the protein molecule to its cognate binding sequence. When such a molecule binds the test sequence the equilibrium of the DNA:protein complexes is disturbed, generating changes in the concentration of free DNA probe. Numerous exemplary target test sequences (SEQ ID NO:1 to SEQ ID NO:600) are set forth. The assay of the present invention is also useful to characterize the preferred binding sequences of any selected DNA-binding molecule.

L10 ANSWER 10 OF 10 CAPLUS COPYRIGHT 2001 ACS
AN 1999:159061 CAPLUS
DN 131:14761
TI Characterization of nra, a global negative regulator gene in group A **streptococci**
AU Podbielski, Andreas; Woischnik, Markus; Leonard, Bettina A. B.; Schmidt, Karl-Hermann
CS Department of Medical Microbiology and Hygiene, University Hospital Ulm, Ulm, D-89081, Germany
SO Mol. Microbiol. (1999), 31(4), 1051-1064
CODEN: MOMIEE; ISSN: 0950-382X
PB Blackwell Science Ltd.
DT Journal
LA English
AB During sequencing of an 11.5 kb genomic region of a serotype M49 group A **streptococcal** (GAS) strain, a series of genes were identified including nra (neg. regulator of GAS). Transcriptional anal. of the region revealed that nra was primarily monocistronically transcribed. Polycistronic expression was found for the three open reading frames (ORFs) downstream and for the four ORFs upstream of nra. The deduced Nra protein sequence exhibited 62% homol. to the GAS RofA pos. regulator. In contrast to RofA, Nra was found to be a neg. regulator of its own expression and that of the two adjacent operons by anal. of insertional inactivation mutants. By polymerase chain reaction and hybridization assays of 10 different GAS serotypes, the genomic presence of nra, rofA or both was demonstrated. Nra-regulated genes include the fibronectin-binding protein F2 gene (prtF2) and a novel **collagen** -binding protein (**cpa**). The **Cpa** polypeptide was purified as a recombinant maltose-binding protein fusion and shown to bind type I **collagen** but not fibronectin. In accordance with nra acting as a neg. regulator of prtF2 and **cpa**, levels of attachment of the nra mutant strain to immobilized **collagen** and fibronectin was increased above wild-type levels. In addn., nra was also found to regulate neg. (four- to 16-fold) the global pos. regulator gene, mga. Using a strain carrying a chromosomally integrated duplication of the nra 3' end and an nra-luciferase reporter gene transcriptional fusion,

nra expression was obsd. to reach its max. during late logarithmic growth phase, while no significant influence of atm. conditions could be distinguished clearly.
RE.CNT 53
RE
(2) Brakhage, A; Biochimie 1990, V72, P725 CAPLUS
(3) Caparon, M; J Bacteriol 1992, V174, P5693 CAPLUS
(4) Caparon, M; Methods Enzymol 1991, V204, P556 CAPLUS
(5) Chen, C; Mol Gen Genet 1993, V241, P685 CAPLUS
(6) Chen, D; J Biol Chem 1994, V269, P32120 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> d his

(FILE 'HOME' ENTERED AT 15:44:35 ON 21 JUN 2001)

FILE 'USPATFULL, WPIDS, CAPLUS, AGRICOLA' ENTERED AT 15:45:05 ON 21 JUN 2001
E PODBIELSKI ANDREAS/AU
L1 83 S E2-E4
L2 1 S L1 AND COLLAGEN
L3 60 S L1 AND STREPTOCOCC?
L4 17 S L3 AND BINDING (5A) PROTEIN
L5 17 DUP REM L4 (0 DUPLICATES REMOVED)
L6 55879 S STREPTOCOCC?
L7 903 S L6 AND COLLAGEN
L8 0 S L7 AND CPA1
L9 10 S L7 AND CPA?
L10 10 DUP REM L9 (0 DUPLICATES REMOVED)

=> s l7 and adhesion

L11 231 L7 AND ADHESION

=> s l11 and collagen (5a) binding

L12 48 L11 AND COLLAGEN (5A) BINDING

=> s l12 and (dna or polynucleotid? or amino acid or protein or polypeptid?)

1 FILES SEARCHED...
3 FILES SEARCHED...
L13 43 L12 AND (DNA OR POLYNUCLEOTID? OR AMINO ACID OR PROTEIN OR POLYP
EPTID?)

=> dup rem l13

PROCESSING COMPLETED FOR L13
L14 43 DUP REM L13 (0 DUPLICATES REMOVED)

=> d bib ab 1-43

L14 ANSWER 1 OF 43 USPATFULL
AN 2001:93284 USPATFULL
TI Decorin binding **protein** compositions and methods of use
IN Guo, Betty P., Boston, MA, United States
Hook, Magnus, Houston, TX, United States
PA The Texas A & M University System, College Station, TX, United States (U.S. corporation)
PI US 6248517 B1 20010619
WO 9634106 19961031

AI US 1997-945476 19971224 (8)
WO 1996-US5886 19960424
19971224 PCT 371 date
19971224 PCT 102(e) date
RLI Continuation-in-part of Ser. No. US 1996-589711, filed on 22 Jan 1996, now patented, Pat. No. US 5853987 Continuation-in-part of Ser. No. US 1995-427023, filed on 24 Apr 1995, now abandoned
DT Utility
EXNAM Primary Examiner: Zitomer, Stephanie W..
LREP Williams, Morgan and Amerson
CLMN Number of Claims: 57
ECL Exemplary Claim: 1
DRWN 42 Drawing Figure(s); 28 Drawing Page(s)
LN.CNT 4945
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are the dbp gene and dbp-derived nucleic acid segments from Borrelia burgdorferi, the etiological agent of Lyme disease, and **DNA** segments encoding dbp from related borrelias. Also disclosed are decorin binding **protein** compositions and methods of use. The DBP **protein** and antigenic epitopes derived therefrom are contemplated for use in the treatment of pathological Borrelia infections, and in particular, for use in the prevention of bacterial **adhesion** to decorin. **DNA** segments encoding these proteins and anti-(decorin binding **protein**) antibodies will also be of use in various screening, diagnostic and therapeutic applications including active and passive immunization and methods for the prevention of Borrelia colonization in an animal. These **DNA** segments and the peptides derived therefrom are contemplated for use in the preparation of vaccines and, also, for use as carrier proteins in vaccine formulations, and in the formulation of compositions for use in the prevention of Lyme disease.

L14 ANSWER 2 OF 43 USPATFULL
AN 2001:86039 USPATFULL
TI Choline binding proteins for anti-pneumococcal vaccines
IN Masure, H. Robert, Germantown, TN, United States
Rosenow, Carsten I., New York, NY, United States
Tuomanen, Elaine, Germantown, TN, United States
Wizemann, Theresa M., Germantown, MD, United States
PA The Rockefeller University, New York, NY, United States (U.S. corporation)
PI US 6245335 B1 20010612
AI US 1997-847065 19970501 (8)
PRAI US 1996-16632 19960501 (60)
DT Utility
EXNAM Primary Examiner: Mosher, Mary E.
LREP Klauber & Jackson
CLMN Number of Claims: 19
ECL Exemplary Claim: 1
DRWN 25 Drawing Figure(s); 18 Drawing Page(s)
LN.CNT 2933
AB The invention relates to bacterial choline binding proteins (CBPs) which bind choline. Such proteins are particularly desirable for vaccines against appropriate strains of Gram positive bacteria, particularly **streptococcus**, and more particularly pneumococcus. Also provided are **DNA** sequences encoding the bacterial choline binding proteins or fragment thereof, antibodies to the bacterial choline binding proteins, pharmaceutical compositions comprising the bacterial choline binding proteins, antibodies to the bacterial choline binding proteins suitable for use in passive immunization, and small molecule inhibitors of choline binding **protein** mediated **adhesion**. Methods for diagnosing the presence of the bacterial choline binding **protein**, or of the bacteria, are also provided. In a specific embodiment, a **streptococcal** choline

binding **protein** is an enolase, which demonstrates strong affinity for fibronectin.

L14 ANSWER 3 OF 43 USPATFULL
AN 2001:67646 USPATFULL
TI Decorin binding **protein** compositions
IN Guo, Betty, Houston, TX, United States
Hook, Magnus, Houston, TX, United States
PA The Texas A & M Unversity System, College Station, TX, United States (U.S. corporation)
PI US 6228835 B1 20010508
AI US 1998-221938 19981228 (9)
RLI Division of Ser. No. US 1996-589711, filed on 22 Jan 1996, now patented,
Pat. No. US 5853987, issued on 29 Dec 1998 Continuation-in-part of Ser. No. US 1995-427023, filed on 24 Apr 1995, now abandoned
DT Utility
EXNAM Primary Examiner: Zitomer, Stephanie W.
LREP Williams, Morgan and Amerson
CLMN Number of Claims: 24
ECL Exemplary Claim: 1
DRWN 25 Drawing Figure(s); 14 Drawing Page(s)
LN.CNT 4504
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are the dbp gene and dbp-derived nucleic acid segments from Borrelia burgdorferi, the etiological agent of Lyme disease, and **DNA** segments encoding dbp from related borrelias. Also disclosed are decorin binding **protein** compositions and methods of use. The DBP **protein** and antigenic epitopes derived therefrom are contemplated for use in the treatment of pathological Borrelia infections, and in particular, for use in the prevention of bacterial **adhesion** to decorin. **DNA** segments encoding these proteins and anti-(decorin binding **protein**) antibodies will also be of use in various screening, diagnostic and therapeutic applications including active and passive immunization and methods for the prevention of Borrelia colonization in an animal. These **DNA** segments and the peptides derived therefrom are contemplated for use in the preparation of vaccines and, also, for use as carrier proteins in vaccine formulations, and in the formulation of compositions for use in the prevention of Lyme disease.

L14 ANSWER 4 OF 43 USPATFULL
AN 2001:59685 USPATFULL
TI Co-cultivation of cells in a micropatterned configuration
IN Bhatia, Sangeeta, Cambridge, MA, United States
Yarmush, Martin, Newton, MA, United States
Toner, Mehmet, Wellesley, MA, United States
PA The General Hospital Corporation Massachusetts Institute of Technology, Cambridge, MA, United States (U.S. corporation)
PI US 6221663 B1 20010424
AI US 2000-482017 20000113 (9)
RLI Division of Ser. No. US 1997-943143, filed on 3 Oct 1997, now patented, Pat. No. US 6133030
DT Utility
EXNAM Primary Examiner: Tate, Christopher R.
LREP Fish & Richardson P.C.
CLMN Number of Claims: 22
ECL Exemplary Claim: 1
DRWN 4 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 1870
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are methods for producing co-cultures of cells in which at least two cell types are present in a micropattern configuration.

L14 ANSWER 5 OF 43 USPATFULL

AN 2001:59420 USPATFULL
TI Surface cross-linked particles suitable for controlled delivery
IN Russell-Jones, Gregory J., Middle Cove, Australia
Starling, Scott M., Bexley, Australia
McEwan, John F., Oatley, Australia
PA Biotech Australia PTY Limited, Roseville, Australia (non-U.S. corporation)
PI US 6221397 B1 20010424
AI US 1998-143118 19980828 (9)
PRAI AU 1997-8880 19970829
DT Utility
EXNAM Primary Examiner: Page, Thurman K.; Assistant Examiner: Seidleck, Brian K.
LREP Foley & Lardner
CLMN Number of Claims: 29
ECL Exemplary Claim: 1
DRWN 5 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 1144
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Cross-linked particles are provided that are useful for delivery of pharmaceutical agents. The particles comprise at least one polymeric compound and a spacer compound, where the polymeric compound and the spacer each comprise reactive carboxyl, hydrazidyl, amino and/or thiol groups. The particles are cross-linked via covalent linkage of the reactive groups on the polymer and spacer respectively. Compositions comprising pharmaceutical agents contained within the particles are disclosed. Methods for preparing the particles, for encapsulating pharmaceutical agents within the particles, and for using the particles for controlled release of the pharmaceutical agent within the patient also are provided.

L14 ANSWER 6 OF 43 USPATFULL
AN 2001:51579 USPATFULL
TI DbpA compositions
IN Guo, Betty P., Boston, MA, United States
Hook, Magnus, Houston, TX, United States
PA Texas A & M University System, College Station, TX, United States (U.S. corporation)
PI US 6214355 B1 20010410
WO 9727301 19970731
AI US 1998-117257 19980722 (9)
WO 1996-US17081 19961022
19981029 PCT 371 date
19981029 PCT 102(e) date
RLI Continuation-in-part of Ser. No. US 945476 Continuation-in-part of Ser. No. US 1996-589711, filed on 22 Jan 1996, now patented, Pat. No. US 5853987, issued on 29 Dec 1998 Continuation-in-part of Ser. No. US 1995-427023, filed on 24 Apr 1995, now abandoned
DT Utility
EXNAM Primary Examiner: Zitomer, Stephanie W.
LREP Williams, Morgan and Amerson
CLMN Number of Claims: 39
ECL Exemplary Claim: 1
DRWN 34 Drawing Figure(s); 31 Drawing Page(s)
LN.CNT 5444
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are the dbp gene and dbp-derived nucleic acid segments from Borrelia burgdorferi, the etiological agent of Lyme disease, and **DNA** segments encoding dbp from related borrelias. Also disclosed are decorin binding **protein** compositions and methods of use. The DBP **protein** and antigenic epitopes derived therefrom are contemplated for use in the treatment of pathological Borrelia infections, and in particular, for use in the prevention of bacterial **adhesion** to decorin. **DN**

PRAI AU 1997-8880 19970829
DT Utility
EXNAM Primary Examiner: Page, Thurman K.; Assistant Examiner: Seidleck, Brian K.
LREP Foley & Lardner
CLMN Number of Claims: 29
ECL Exemplary Claim: 1
DRWN 5 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 1144
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Cross-linked particles are provided that are useful for delivery of pharmaceutical agents. The particles comprise at least one polymeric compound and a spacer compound, where the polymeric compound and the spacer each comprise reactive carboxyl, hydrazidyl, amino and/or thiol groups. The particles are cross-linked via covalent linkage of the reactive groups on the polymer and spacer respectively. Compositions comprising pharmaceutical agents contained within the particles are disclosed. Methods for preparing the particles, for encapsulating pharmaceutical agents within the particles, and for using the particles for controlled release of the pharmaceutical agent within the patient also are provided.

L14 ANSWER 6 OF 43 USPATFULL
AN 2001:51579 USPATFULL
TI DbpA compositions
IN Guo, Betty P., Boston, MA, United States
Hook, Magnus, Houston, TX, United States
PA Texas A & M University System, College Station, TX, United States (U.S. corporation)
PI US 6214355 B1 20010410
WO 9727301 19970731
AI US 1998-117257 19980722 (9)
WO 1996-US17081 19961022
19981029 PCT 371 date
19981029 PCT 102(e) date
RLI Continuation-in-part of Ser. No. US 945476 Continuation-in-part of Ser. No. US 1996-589711, filed on 22 Jan 1996, now patented, Pat. No. US 5853987, issued on 29 Dec 1998 Continuation-in-part of Ser. No. US 1995-427023, filed on 24 Apr 1995, now abandoned
DT Utility
EXNAM Primary Examiner: Zitomer, Stephanie W.
LREP Williams, Morgan and Amerson
CLMN Number of Claims: 39
ECL Exemplary Claim: 1
DRWN 34 Drawing Figure(s); 31 Drawing Page(s)

LN.CNT 5444
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are the dbp gene and dbp-derived nucleic acid segments from Borrelia burgdorferi, the etiological agent of Lyme disease, and ***DNA*** segments encoding dbp from related borrelias. Also disclosed are decorin binding ***protein*** compositions and methods of use. The DBP ***protein*** and antigenic epitopes derived therefrom are contemplated for use in the treatment of pathological Borrelia infections, and in particular, for use in the prevention of bacterial ***adhesion*** to decorin. ***DNA*** segments encoding these proteins and anti-(decorin binding ***protein***) antibodies will also be of use in various screening, diagnostic and therapeutic applications including active and passive immunization and methods for the prevention of Borrelia colonization in an animal. These ***DNA*** segments and the peptides derived therefrom are contemplated for use in the preparation of vaccines and, also, for use as carrier proteins in vaccine formulations, and in the formulation of compositions for use in the prevention of Lyme disease.

L14 ANSWER 7 OF 43 USPATFULL
AN 2001:22438 USPATFULL
TI Transgenic animals as model of psoriasis
IN Watt, Fiona M., London, United Kingdom
Carroll, Joseph M., London, United Kingdom
PA Imperial Cancer Research Technology Limited, London, United Kingdom (non-U.S. corporation)
PI US 6187993 B1 20010213
WO 9627019 19960906
AI US 1997-894649 19971103 (8)
WO 1996-GB431 19960226
19971103 PCT 371 date
19971103 PCT 102(e) date
PRAI GB 1995-9603868 19950225
GB 1995-14535 19950715
DT Utility
EXNAM Primary Examiner: Priebe, Scott D.; Assistant Examiner: Baker, Anne-Marie
LREP Nixon & Vanderhyde P.C.
CLMN Number of Claims: 14
ECL Exemplary Claim: 1
DRWN 17 Drawing Figure(s); 17 Drawing Page(s)
LN.CNT 1960
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A nucleic acid construct comprising a promoter capable of directing expression in the suprabasal cells of the epidermis and means to cause

expression of an integrin subunit in the suprabasal cells. Preferably the means to cause expression of an integrin subunit is an integrin subunit coding sequence. A transgenic animal which expresses an .alpha. subunit and a .beta. subunit of integrin in the suprabasal cells of the epidermis and methods for making the transgenic animals. At least some of the transgenic animals are useful models of human disease, especially psoriasis. A method of treating psoriasis comprising administering to the patient a compound which modulates integrin function.

L14 ANSWER 8 OF 43 USPATFULL
AN 2001:10546 USPATFULL
TI S. aureusfibrinogen binding ***protein***
IN Foster, Timothy James, Dublin, Ireland
McDevitt, Damien Leo, Dublin, Ireland
PA The Provost, Fellows and Scholars of The College of the Holy and Undivided Trinity of Queen Elizabeth Near Dublin, Dublin, Ireland (non-U.S. corporation)
PI US 6177084 B1 20010123
AI US 1999-421868 19991019 (9)
RLI Division of Ser. No. US 1994-293728, filed on 22 Aug 1994, now patented, Pat. No. US 6008341
DT Utility
EXNAM Primary Examiner: Graser, Jennifer
LREP Larson & Taylor PLC
CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN 13 Drawing Figure(s); 13 Drawing Page(s)
LN.CNT 812
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The isolation of the S. aureus fibrinogen binding ***protein*** gene is described and a minimal fibrinogen binding ***protein*** is identified. The ***protein*** finds use as a vaccine or a pharmaceutical composition for application to prevent infection, promotion of wound healing, blocking adherence to indwelling medical devices, or diagnosis of infection.

L14 ANSWER 9 OF 43 USPATFULL
AN 2000:164486 USPATFULL
TI Anti-fungal peptides
IN Little, II, Roger G., Benicia, CA, United States
Lim, Edward, Walnut Creek, CA, United States
Fadem, Mitchell B., Berkeley, CA, United States
PA Xoma Corporation, Berkeley, CA, United States (U.S. corporation)
PI US 6156730 20001205
AI US 1999-227659 19990108 (9)

RLI Continuation of Ser. No. US 1996-621259, filed on 21 Mar 1996, now patented, Pat. No. US 5858974 which is a continuation-in-part of Ser. No. US 1995-504841, filed on 20 Jul 1995, now abandoned which is a continuation-in-part of Ser. No. US 1995-372105, filed on 13 Jan 1995, now patented, Pat. No. US 5627153 which is a continuation-in-part of Ser. No. US 1994-306473, filed on 15 Sep 1994, now patented, Pat. No. US 5652332 And a continuation-in-part of Ser. No. US 1994-273540, filed on 11 Jul 1994, now abandoned which is a continuation-in-part of Ser. No. US 1994-209762, filed on 11 Mar 1994, now patented, Pat. No. US 5733872 which is a continuation-in-part of Ser. No. US 1994-183222, filed on 14 Jan 1994, now abandoned which is a continuation-in-part of Ser. No. US 1993-93202, filed on 15 Jul 1993, now abandoned which is a continuation-in-part of Ser. No. US 1993-30644, filed on 12 Mar 1993, now patented, Pat. No. US 5348942
DT Utility
EXNAM Primary Examiner: Davenport, Avis M.
LREP McAndrews, Held & Malloy, Ltd.
CLMN Number of Claims: 15
ECL Exemplary Claim: 1
DRWN 9 Drawing Figure(s); 10 Drawing Page(s)
LN.CNT 6157
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates generally to anti-fungal peptides derived from or based on Domain III (amino acids 142-169) of bactericidal/permeability-increasing ***protein*** (BPI) and in vivo or in vitro uses of such peptides.

L14 ANSWER 10 OF 43 USPATFULL
AN 2000:138120 USPATFULL
TI Co-cultivation of cells in a micropatterned configuration
IN Bhatia, Sangeeta, Cambridge, MA, United States
Yarmush, Martin, Newton, MA, United States
Toner, Mehmet, Wellesley, MA, United States
PA The General Hospital Corporation, Boston, MA, United States (U.S. corporation)
Massachusetts Institute of Technology, Cambridge, MA, United States (U.S. corporation)
PI US 6133030 20001017
AI US 1997-943143 19971003 (8)
DT Utility
EXNAM Primary Examiner: Lankford, Jr., Leon B.; Assistant Examiner: Tate, Christopher R.
LREP Fish & Richardson P.C.
CLMN Number of Claims: 25
ECL Exemplary Claim: 1

DRWN 3 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 1851
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are methods for producing co-cultures of cells in which at least two cell types are present in a micropattern configuration.

L14 ANSWER 11 OF 43 USPATFULL
AN 2000:87726 USPATFULL
TI Fibronectin binding ***protein***
IN Hook, Magnus, Birmingham, AL, United States
Lindberg, Kjell Martin, Uppsala, Sweden
Lindgren, Per-Eric, Uppsala, Sweden
Signas, Lars Christer, Uppsala, Sweden
PA Alfa Laval Agri International, Sweden (non-U.S. corporation)
PI US 6086895 20000711
AI US 1997-904179 19970801 (8)
RLI Continuation of Ser. No. US 1995-428713, filed on 25 Apr 1995, now patented, Pat. No. US 5866541 which is a division of Ser. No. US 1993-125222, filed on 23 Sep 1993, now patented, Pat. No. US 5416021 which is a continuation of Ser. No. US 1992-973551, filed on 9 Nov 1992, now abandoned which is a continuation of Ser. No. US 1989-352949, filed on 17 May 1989, now abandoned
PRAI SE 1998-8801894 19980520
DT Utility
EXNAM Primary Examiner: Minnifield, Nita
LREP Burns, Doane, Swecker, & Mathis, L.L.P.
CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN 5 Drawing Figure(s); 9 Drawing Page(s)
LN.CNT 918
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to new recombinant ***DNA*** -molecules comprising nucleotide sequences of S. dygalactiae encoding for at least one ***protein*** or ***polypeptide*** having fibronectin binding property.

L14 ANSWER 12 OF 43 USPATFULL
AN 2000:24479 USPATFULL
TI Fibronectin binding ***protein*** as well as its preparation
IN Normark, Staffan, S-913 00 Holmsund, Zackrisvagen, Sweden
Olsen, Arne, 902 41 Umea, Sprakgrand 19, Sweden
PI US 6030805 20000229
AI US 1995-495959 19950628 (8)
RLI Continuation of Ser. No. US 1994-318519, filed on 5 Oct 1994, now abandoned which is a continuation of Ser. No. US 1994-187865, filed on

28 Jan 1994, now abandoned which is a continuation of Ser. No. US 1992-970846, filed on 3 Nov 1992, now abandoned which is a continuation-in-part of Ser. No. US 1991-789437, filed on 6 Nov 1991, now abandoned which is a continuation of Ser. No. US 1989-347189, filed on 4 May 1989, now abandoned
DT Utility
EXNAM Primary Examiner: Wax, Robert A.; Assistant Examiner: Mytelka, Daniel S.
LREP Burns, Doane, Swecker & Mathis, L.L.P.
CLMN Number of Claims: 17
ECL Exemplary Claim: 1
DRWN 7 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 715
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to a new fibronectin binding ***protein*** from E. coli in the form of a curli pili, a new recombinant hybrid- ***DNA*** -molecule comprising a nucleotide sequence from E. coli coding for a ***protein*** or ***polypeptide*** having fibronectin binding properties. (FIG. 4).

L14 ANSWER 13 OF 43 USPATFULL
AN 2000:4645 USPATFULL
TI Cell surface ***protein*** compounds
IN Hodgson, John Edward, Malvern, PA, United States
Burnham, Martin Karl Russell, Norristown, PA, United States
PA SmithKline Beecham plc, United Kingdom (non-U.S. corporation)
PI US 6013482 20000111
AI US 1996-730261 19961015 (8)
DT Utility
EXNAM Primary Examiner: Caputa, Anthony C.; Assistant Examiner: Navarro, Mark
LREP Gimmi, Edward R., King, William T., Deibert, Thomas S.
CLMN Number of Claims: 14
ECL Exemplary Claim: 1
DRWN 5 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 1255
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel cell surface ***protein*** ***polypeptides*** and ***DNA*** (RNA) encoding such novel cell surface ***protein*** and a procedure for producing such ***polypeptides*** by recombinant techniques is disclosed. Also disclosed are methods for utilizing such novel cell surface ***protein*** for the treatment of infection, particularly bacterial infections. Antagonists against such novel cell surface ***protein*** and their use as a therapeutic to treat infections, particularly bacterial infections are also disclosed. Also disclosed are diagnostic assays for detecting diseases related to the presence of novel cell surface ***protein*** nucleic acid sequences

and the ***polypeptides*** in a host. Also disclosed are diagnostic assays for detecting ***polynucleotides*** encoding cell surface **protein*** family and for detecting the ***polypeptide** in a host.

L14 ANSWER 14 OF 43 CAPLUS COPYRIGHT 2001 ACS
AN 2000:146804 CAPLUS
DN 132:277575
TI Coinvasion of dentinal tubules by Porphyromonas gingivalis and ***Streptococcus*** gordonii depends upon binding specificity of ***streptococcal*** antigen I/II adhesin
AU Love, Robert M.; McMillan, Malcolm D.; Park, Yoonsuk; Jenkinson, Howard F.
CS School of Dentistry, University of Otago, Dunedin, N. Z.
SO Infect. Immun. (2000), 68(3), 1359-1365
CODEN: INFIBR; ISSN: 0019-9567
PB American Society for Microbiology
DT Journal
LA English
AB Cell wall-anchored ***polypeptides*** of the antigen I/II family are produced by many species of oral ***streptococci*** . These proteins mediate ***adhesion*** of ***streptococci*** to salivary glycoproteins and to other oral microorganisms and promote ***binding*** of cells to ***collagen*** type I and invasion of dentinal tubules. Since infections of the root canal system have a mixed anaerobic bacterial etiol., the authors investigated the hypothesis that coadhesion of anaerobic bacteria with ***streptococci*** may facilitate invasive endodontic disease. Porphyromonas gingivalis ATCC 33277 cells were able to invade dentinal tubules when cocultured with ***Streptococcus*** gordonii DL1 (Challis) but not when cocultured with ***Streptococcus*** mutans NG8. An isogenic noninvasive mutant of S. gordonii, with prodn. of SspA and SspB (antigen I/II family) ***polypeptides*** abrogated, was deficient in ***binding*** to ***collagen*** and had a 40% reduced ability to support ***adhesion*** of P. gingivalis. Heterologous expression of the S. mutans SpaP (antigen I/II) ***protein*** in this mutant restored ***collagen*** ***binding*** and tubule invasion but not ***adhesion*** to P. gingivalis or the ability to promote P. gingivalis coinvasion of dentin. An isogenic afimbrial mutant of P. gingivalis had 50% reduced binding to S. gordonii cells but was unaffected in the ability to coinvade dentinal tubules with S. gordonii wild-type cells. Expression of the S. gordonii SspA or SspB ***polypeptide*** on the surface of Lactococcus lactis cells endowed these bacteria with the abilities to bind P. gingivalis, penetrate dentinal tubules, and promote P. gingivalis coinvasion of dentin. The results demonstrate that ***collagen*** - ***binding*** and P. gingivalis- ***binding*** properties of antigen I/II ***polypeptides*** are discrete functions.

Specificity of antigen I/II ***polypeptide*** recognition accounts for the ability of P. gingivalis to coinvade dentinal tubules with S. gordonii but not with S. mutans. This provides evidence that the specificity of interbacterial coadhesion may influence directly the etiol. of pulpal and periapical diseases.

RE.CNT 47

RE

(4) Bradshaw, D; Infect Immun 1998, V66, P4729 CAPLUS

(5) Brooks, W; Infect Immun 1997, V65, P3753 CAPLUS

(8) Crowley, P; Infect Immun 1999, V67, P1201 CAPLUS

(9) Dai, X; Arch Oral Biol 1991, V36, P775 CAPLUS

(10) Demuth, D; Mol Microbiol 1996, V20, P403 CAPLUS

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 15 OF 43 CAPLUS COPYRIGHT 2001 ACS

AN 2001:8445 CAPLUS

DN 134:191510

TI Co-operative binding of human fibronectin to SfbI ***protein*** triggers ***streptococcal*** invasion into respiratory epithelial cells

AU Talay, Susanne R.; Zock, Angela; Rohde, Manfred; Molinari, Gabriella; Oggioni, Marco; Pozzi, Gianni; Guzman, Carlos A.; Chhatwal, Gursharan S.

CS Division of Microbiology, Technical University/GBF-National Research Centre for Biotechnology, Braunschweig, 38124, Germany

SO Cell. Microbiol. (2000), 2(6), 521-535
CODEN: CEMIF5; ISSN: 1462-5814

PB Blackwell Science Ltd.

DT Journal

LA English

AB ***Streptococcal*** fibronectin binding ***protein*** I (SfbI) mediates adherence to and invasion of ***Streptococcus*** pyogenes into human epithelial cells. In this study, the authors analyzed the binding activity of distinct domains of SfbI ***protein*** towards its ligand, the extracellular matrix component fibronectin, as well as the biol. implication of the binding events during the infection process. By using purified recombinant SfbI derivs. as well as in vivo expressed SfbI domains on the surface of heterologous organism ***Streptococcus*** gordonii, the authors were able to dissoc. the two major ***streptococcal*** target domains on the human fibronectin mol. The SfbI repeat region exclusively bound to the 30 kDa N-terminal fragment of fibronectin, whereas the SfbI spacer region exclusively bound to the 45 kDa ***collagen*** - ***binding*** fragment of fibronectin. In the case of native surface-expressed SfbI ***protein*** , an induced fit mode of bacteria-fibronectin interaction was identified. The authors demonstrate that binding of the 30 kDa fibronectin fragment to the repeat

region of SfbI ***protein*** co-operatively activates the adjacent SfbI spacer domain to bind the 45 kDa fibronectin fragment. The biol. consequence arising from this novel mode of fibronectin targeting was analyzed in eukaryotic cell invasion assays. The repeat region of SfbI ***protein*** is mediating adherence and constitutes a prerequisite for subsequent invasion, whereas the SfbI spacer domain efficiently triggers the invasion process of ***streptococci*** into the eukaryotic cell. Thus, the authors were able to dissect bacterial ***adhesion*** from invasion by manipulating one ***protein*** . SfbI ***protein*** therefore represents a highly evolved prokaryotic mol. that exploits the host factor fibronectin not only for extracellular targeting but also for its subsequent activation that leads to efficient cellular invasion.

RE.CNT 52

RE

(1) Barkalow, F; J Biol Chem 1991, V266, P7812 CAPLUS

(3) Blaszczak, A; EMBO J 1995, V14, P5085 CAPLUS

(4) Courtney, H; Infect Immun 1994, V62, P3937 CAPLUS

(5) Cue, D; Infect Immun 1998, V66, P4593 CAPLUS

(6) Dersch, P; EMBO J 1999, V18, P1199 CAPLUS

ALL CITATIONS AVAILABLE IN THE RE FORMAT

L14 ANSWER 16 OF 43 USPATFULL

AN 1999:170733 USPATFULL

TI S. aureus fibrinogen binding ***protein*** gene

IN Foster, Timothy James, Dublin, Ireland
McDevitt, Damien Leo, Dublin, Ireland

PA The Provost, Fellows and Scholars of the College of the Holy and Undivided Trinity of Queen Elizabeth Near Dublin, Dublin, Ireland (non-U.S. corporation)

PI US 6008341 19991228

AI US 1994-293728 19940822 (8)

DT Utility

EXNAM Primary Examiner: Chin, Christopher L.; Assistant Examiner: Graser, Jennifer

LREP Larson & Taylor

CLMN Number of Claims: 12

ECL Exemplary Claim: 1

DRWN 19 Drawing Figure(s); 16 Drawing Page(s)

LN.CNT 1493

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The isolation of the S. aureus fibrinogen binding ***protein*** gene is described and a minimal fibrinogen binding ***protein*** is identified. The ***protein*** finds use as a vaccine or a pharmaceutical composition for application to prevent infection, promotion of wound healing, blocking adherence to indwelling medical

devices, or diagnosis of infection.

L14 ANSWER 17 OF 43 USPATFULL
AN 1999:65197 USPATFULL
TI ***DNA*** encoding fibronectin and fibrinogen binding ***protein*** from group A ***streptococci***
IN Rocha, Claudia, New York, NY, United States
Fischetti, Vincent A., West Hempstead, NY, United States
PA The Rockefeller University, New York, NY, United States (U.S. corporation)
PI US 5910441 19990608
AI US 1996-714402 19960916 (8)
DT Utility
EXNAM Primary Examiner: Caputa, Anthony C.; Assistant Examiner: Navarro, Mark
LREP Burns, Doane, Swecker & Mathis, L.L.P.
CLMN Number of Claims: 11
ECL Exemplary Claim: 1
DRWN 5 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 959
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB This invention relates to a novel fibrinogen and fibronectin binding ***protein*** from group A ***streptococci*** , and the ***DNA*** encoding the ***protein*** . The ***protein*** and its ***DNA*** are useful in the preparation of compositions for the diagnosis, treatment, and prevention of ***streptococcal*** infection.

L14 ANSWER 18 OF 43 USPATFULL
AN 1999:33556 USPATFULL
TI Lep
IN Lonetto, Michael Arthur, Collegeville, PA, United States
PA SmithKline Beecham Corporation, Philadelphia, PA, United States (U.S. corporation)
PI US 5882643 19990316
AI US 1997-964494 19971105 (8)
RLI Division of Ser. No. US 1996-756299, filed on 25 Nov 1996, now patented, Pat. No. US 5786197
DT Utility
EXNAM Primary Examiner: Wax, Robert A.; Assistant Examiner: Slobodyansky, Elizabeth
LREP Gimmi, Edward R., King, William T., Jackson, Arthur E.
CLMN Number of Claims: 3
ECL Exemplary Claim: 1
DRWN 3 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 2053

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB lep ***polypeptides*** and ***DNA ** (RNA) encoding such lep and a procedure for producing such ***polypeptides*** by recombinant techniques is disclosed. Also disclosed are methods for utilizing such lep for the treatment of infection, particularly bacterial infections. Antagonists against such lep and their use as a therapeutic to treat infections, particularly bacterial infections are also disclosed. Also disclosed are diagnostic assays for detecting diseases related to the presence of lep nucleic acid sequences and the ***polypeptides*** in a host. Also disclosed are diagnostic assays for detecting ***polynucleotides*** encoding leader peptidase and for detecting the ***polypeptide*** in a host.

L14 ANSWER 19 OF 43 USPATFULL

AN 1999:15895 USPATFULL

TI Fibronection binding ***protein*** from ***Streptococcus*** dysgalactiae

IN Hook, Magnus, Birmingham, AL, United States
Lindberg, Kjell Martin, Upsala, Sweden
Lindgren, Per-Eric, Upsala, Sweden
Signas, Lars Christer, Upsala, Sweden

PA Alfa-Laval Agri International Aktiebolag, Tumba, Sweden (non-U.S. corporation)

PI US 5866541 19990202

AI US 1995-428713 19950425 (8)

RLI Division of Ser. No. US 1993-125222, filed on 23 Sep 1993, now patented, Pat. No. US 5416021 which is a continuation of Ser. No. US 1992-973551, filed on 9 Nov 1992, now abandoned which is a continuation of Ser. No. US 1989-352949, filed on 17 May 1989, now abandoned

PRAI SE 1988-1894 19880520

DT Utility

EXNAM Primary Examiner: Walsh, Stephen; Assistant Examiner: Brown, Karen E.

LREP Burns, Doane, Swecker & Mathis

CLMN Number of Claims: 10

ECL Exemplary Claim: 1

DRWN 10 Drawing Figure(s); 8 Drawing Page(s)

LN.CNT 1126

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to new recombinant ***DNA*** -molecules comprising nucleotide sequences of S. dysgalactiae encoding for at least one ***protein*** or ***polypeptide*** having fibronectin binding property.

L14 ANSWER 20 OF 43 USPATFULL

AN 1999:4632 USPATFULL

TI Anti-fungal peptides
IN Little, II, Roger G., Benicia, CA, United States
Lim, Edward, Walnut Creek, CA, United States
Fadem, Mitchell B., Carmel Valley, CA, United States
PA XOMA Corporation, Berkeley, CA, United States (U.S. corporation)
PI US 5858974 19990112
AI US 1996-621259 19960321 (8)
RLI Continuation-in-part of Ser. No. US 1995-504841, filed on 20 Jul 1995
DT Utility
EXNAM Primary Examiner: Davenport, Avis M.
LREP McAndrews, Held & Malloy, Ltd.
CLMN Number of Claims: 15
ECL Exemplary Claim: 1
DRWN 10 Drawing Figure(s); 10 Drawing Page(s)
LN.CNT 5315
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates generally to anti-fungal peptides derived from or based on Domain III (amino aids 142-169) of bactericidal/permeability-increasing ***protein*** (BPI) and in vivo or in vitro uses of such peptides.

L14 ANSWER 21 OF 43 USPATFULL
AN 1999:4374 USPATFULL
TI Fibronectin binding ***protein*** B compounds
IN Hodgson, John Edward, Malvern, PA, United States
Burnham, Martin Karl Russell, Norristown, PA, United States
PA SmithKline Beecham p.l.c., United Kingdom (non-U.S. corporation)
PI US 5858709 19990112
AI US 1996-732791 19961015 (8)
DT Utility
EXNAM Primary Examiner: Feisee, Lila; Assistant Examiner: Davis, Minh-Tam
LREP Gimmi, Edward R., King, William T., Jackson, Arthur E.
CLMN Number of Claims: 11
ECL Exemplary Claim: 4
DRWN 3 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 1001
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Novel fibronectin binding ***protein*** B ***polypeptides*** and ***DNA*** (RNA) encoding such novel fibronectin binding ***protein*** B and a procedure for producing such ***polypeptides*** by recombinant techniques is disclosed. Also disclosed are methods for utilizing such novel fibronectin binding ***protein*** B for the treatment of infection, particularly bacterial infections. Antagonists against such novel fibronectin binding ***protein*** B and their use as a therapeutic to treat infections,

particularly bacterial infections are also disclosed. Also disclosed are diagnostic assays for detecting diseases related to the presence of novel fibronectin binding ***protein*** B nucleic acid sequences and the ***polypeptides*** in a host. Also disclosed are diagnostic assays for detecting ***polynucleotides*** encoding novel fibronectin binding ***protein*** B family and for detecting the ***polypeptide*** in a host.

L14 ANSWER 22 OF 43 USPATFULL
AN 1998:162259 USPATFULL
TI Decorin binding ***protein*** compositions and methods of use
IN Guo, Betty, Houston, TX, United States
Hook, Magnus, Houston, TX, United States
PA The Texas A & M University System, College Station, TX, United States (U.S. corporation)
PI US 5853987 19981229
AI US 1996-589711 19960122 (8)
RLI Continuation-in-part of Ser. No. US 1995-427023, filed on 24 Apr 1995, now abandoned
DT Utility
EXNAM Primary Examiner: Horlick, Kenneth R.; Assistant Examiner: Tung, Joyce
LREP Arnold, White & Durkee
CLMN Number of Claims: 68
ECL Exemplary Claim: 1
DRWN 25 Drawing Figure(s); 14 Drawing Page(s)
LN.CNT 4684
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Disclosed are the dbp gene and dbp-derived nucleic acid segments from Borrelia burgdorferi, the etiological agent of Lyme disease, and ***DNA*** segments encoding dbp from related borrelias. Also disclosed are decorin binding ***protein*** compositions and methods of use. The DBP ***protein*** and antigenic epitopes derived therefrom are contemplated for use in the treatment of pathological Borrelia infections, and in particular, for use in the prevention of bacterial ***adhesion*** to decorin. ***DNA*** segments encoding these proteins and anti-(decorin binding ***protein***) antibodies will also be of use in various screening, diagnostic and therapeutic applications including active and passive immunization and methods for the prevention of Borrelia colonization in an animal. These ***DNA*** segments and the peptides derived therefrom are contemplated for use in the preparation of vaccines and, also, for use as carrier proteins in vaccine formulations, and in the formulation of compositions for use in the prevention of Lyme disease.

L14 ANSWER 23 OF 43 USPATFULL

AN 1998:159728 USPATFULL
TI ***Collagen*** * *binding*** ***protein*** as well as its preparation
IN Guss, Bengt, Uppsala, Sweden
Hook, Magnus, Birmingham, AL, United States
Jonsson, Hans, Uppsala, Sweden
Lindberg, Martin, Uppsala, Sweden
Patti, Joseph, Birmingham, AL, United States
Signas, Christer, Uppsala, Sweden
Switalski, Lech, Birmingham, AL, United States
PA Alfa Laval AB, Tumba, Sweden (non-U.S. corporation)
PI US 5851794 19981222
AI US 1995-447031 19950522 (8)
RLI Continuation of Ser. No. US 1992-861804, filed on 21 Aug 1992, now abandoned
PRAI SE 1990-3374 19901022
DT Utility
EXNAM Primary Examiner: Wax, Robert A.; Assistant Examiner: Slobodyansky, Elizabeth
LREP Burns, Doane, Swecker & Mathis
CLMN Number of Claims: 9
ECL Exemplary Claim: 1
DRWN 17 Drawing Figure(s); 14 Drawing Page(s)
LN.CNT 1233
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to a new recombinant ***DNA*** -molecule comprising a nucleotide sequence from S. aureus coding for a ***protein*** , or ***polypeptide*** , having ***collagen*** ***binding*** properties.

L14 ANSWER 24 OF 43 USPATFULL
AN 1998:150802 USPATFULL
TI Technique for the prevention of false positive reactions in immunological testing due to C.sub.1 and C.sub.1q components of the complement and method for screening for rheumatic factor
IN Singer, Jacques, Delray Beach, FL, United States
PA Montefiore Medical Center, Bronx, NY, United States (U.S. corporation)
PI US 5843794 19981201
AI US 1995-564895 19951129 (8)
RLI Continuation of Ser. No. US 1993-14549, filed on 8 Feb 1993, now abandoned which is a continuation-in-part of Ser. No. US 1992-857764, filed on 26 Mar 1992, now abandoned
DT Utility
EXNAM Primary Examiner: Spiegel, Carol A.
LREP Hedman, Gibson & Costigan, P.C.

CLMN Number of Claims: 12
ECL Exemplary Claim: 1
DRWN 3 Drawing Figure(s); 3 Drawing Page(s)
LN.CNT 1280
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A novel technique is disclosed for the prevention of false positive reactions in immunological testing which are caused by interference of C.sub.1 and C.sub.1q. The method is based on heating a sample of a body fluid at a temperature of 59.degree.-64.degree. C. in the presence of a particular neutral salt. A method for screening for rheumatoid factor is also disclosed.

L14 ANSWER 25 OF 43 USPATFULL
AN 1998:147559 USPATFULL
TI Fibronectin binding ***protein*** as well as its preparation
IN Hook, Magnus, Birmingham, AL, United States
Jonsson, Klas, Upsala, Sweden
Lindberg, Kjell Martin, Upsala, Sweden
Signas, Christer, Upsala, Sweden
PA Alfa-Laval Agri International Aktiebolag, Tumba, Sweden (non-U.S. corporation)
PI US 5840846 19981124
AI US 1996-725492 19961004 (8)
RLI Division of Ser. No. US 1994-340458, filed on 14 Nov 1994, now patented, Pat. No. US 5320951 which is a continuation of Ser. No. US 1992-974181, filed on 10 Nov 1992, now abandoned which is a division of Ser. No. US 1990-520808, filed on 9 May 1990, now patented, Pat. No. US 5175096
PRAI SE 1989-1687 19890511
DT Utility
EXNAM Primary Examiner: Low, Christopher S.F.
LREP Burns, Doane, Swecker & Mathis, L.L.P.
CLMN Number of Claims: 8
ECL Exemplary Claim: 1
DRWN 8 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 529
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to a new recombinant hybrid- ***DNA*** -molecule comprising a nucleotide sequence from S. aureus coding for a ***protein*** , or ***polypeptide*** , having fibronectin binding properties.

L14 ANSWER 26 OF 43 USPATFULL
AN 1998:104804 USPATFULL
TI ***Polynucleotide*** encoding saliva binding ***protein***
IN Hodgson, John Edward, Malvern, PA, United States

Burnham, Martin Karl Russell, Norristown, PA, United States
PA SmithKline Beecham p.l.c., Brentford, England (non-U.S. corporation)
PI US 5801234 19980901
AI US 1997-896371 19970718 (8)
RLI Division of Ser. No. US 1996-729202, filed on 15 Oct 1996, now patented, Pat. No. US 5700928
PRAI GB 1995-21147 19951016
GB 1996-4599 19960304
GB 1996-16136 19960801
DT Utility
EXNAM Primary Examiner: Achutamurthy, Ponnathapura; Assistant Examiner: Bui, Phuong T.
LREP Gimmi, Edward R., King, William T., Lentz, Edward T.
CLMN Number of Claims: 20
ECL Exemplary Claim: 1
DRWN 4 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 1286
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Saliva binding ***protein*** ***polypeptides*** and ***DNA*** (RNA) of Staphylococcus aureus encoding such saliva binding ***protein*** and a procedure for producing such ***polypeptides*** by recombinant techniques is disclosed. Also disclosed are methods for utilizing such saliva binding ***protein*** for the treatment of infection, particularly bacterial infections. Antagonists against such saliva binding ***protein*** and their use as a therapeutic to treat infections, particularly bacterial infections are also disclosed. Also disclosed are diagnostic assays for detecting diseases related to the presence of saliva binding ***protein*** nucleic acid sequences and the ***polypeptides*** in a host. Also disclosed are diagnostic assays for detecting ***polynucleotides*** encoding saliva binding ***protein*** family and for detecting the ***polypeptide*** in host.

L14 ANSWER 27 OF 43 USPATFULL
AN 1998:92165 USPATFULL
TI Fibronectin binding ***protein***
IN Hook, Magnus, Birmingham, AL, United States
Lindberg, Kjell Martin, Uppsala, Sweden
Lindgren, Per-Eric, Uppsala, Sweden
Signas, Lars Christer, Uppsala, Sweden
PA Alfa Laval Agri International Aktiebolag, Tumba, Sweden (non-U.S. corporation)
PI US 5789549 19980804
AI US 1996-729766 19961007 (8)
RLI Division of Ser. No. US 1995-428713, filed on 25 Apr 1995 which is a

division of Ser. No. US 1993-125222, filed on 23 Sep 1993, now patented, Pat. No. US 5416021 which is a continuation of Ser. No. US 1992-973551, filed on 9 Nov 1992, now abandoned which is a continuation of Ser. No. US 1989-352949, filed on 17 May 1989, now abandoned
PRAI SE 1988-1894 19880520
DT Utility
EXNAM Primary Examiner: Hutzell, Paula K.; Assistant Examiner: Masood, Khalid
LREP Burns, Doane, Swecker & Mathis, L.L.P.
CLMN Number of Claims: 13
ECL Exemplary Claim: 1
DRWN 7 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 455
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to new recombinant ***DNA*** -molecules comprising nucleotide sequences of S. dysgalactiae encoding for at least one ***protein*** or ***polypeptide*** having fibronectin binding property.

L14 ANSWER 28 OF 43 USPATFULL
AN 1998:88688 USPATFULL
TI lep
IN Lonetto, Michael Arthur, Collegeville, PA, United States
PA SmithKline Beecham Corporation, Philadelphia, PA, United States (U.S. corporation)
PI US 5786197 19980728
AI US 1996-756299 19961125 (8)
DT Utility
EXNAM Primary Examiner: Wax, Robert A.; Assistant Examiner: Slobodyamsky, Elizabeth
LREP Gimmi, Edward R., King, William T., Lentz, Edward T.
CLMN Number of Claims: 35
ECL Exemplary Claim: 1
DRWN 5 Drawing Figure(s); 5 Drawing Page(s)
LN.CNT 2166
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB lep ***polypeptides*** and ***DNA*** (RNA) encoding such lep and a procedure for producing such ***polypeptides*** by recombinant techniques is disclosed. Also disclosed are methods for utilizing such lep for the treatment of infection, particularly bacterial infections. Antagonists against such lep and their use as a therapeutic to treat infections, particularly bacterial infections are also disclosed. Also disclosed are diagnostic assays for detecting diseases related to the presence of lep nucleic acid sequences and the ***polypeptides*** in a host. Also disclosed are diagnostic assays for detecting ***polynucleotides*** encoding leader peptidase and for detecting the

polypeptide in a host.

L14 ANSWER 29 OF 43 CAPLUS COPYRIGHT 2001 ACS

AN 1998:649589 CAPLUS

DN 129:341523

TI Binding properties of ***Streptococcus*** gordonii SspA and SspB (antigen I/II family) ***polypeptides*** expressed on the cell surface of Lactococcus lactis MG1363

AU Holmes, Ann R.; Gilbert, Christophe; Wells, Jeremy M.; Jenkinson, Howard F.

CS Department of Oral Sciences and Orthodontics, University of Otago, Dunedin, N. Z.

SO Infect. Immun. (1998), 66(10), 4633-4639
CODEN: INFIBR; ISSN: 0019-9567

PB American Society for Microbiology

DT Journal

LA English

AB The oral bacterium ***Streptococcus*** gordonii expresses two cell wall-assocd. ***polypeptides*** , designated SspA (1,542 ***amino*** ***acid*** residues) and SspB (1,462 ***amino*** ***acid*** residues), that have 70% sequence identity. These ***polypeptides*** are members of the antigen I/II family of oral ***streptococcal*** adhesins and mediate the ***binding*** of ***streptococci*** to salivary glycoproteins, ***collagen*** , and other oral microorganisms such as Actinomyces naeslundii. To det. if SspA and SspB have differential binding properties, the coding sequences of the sspA and sspB genes were cloned into expression plasmid vector pTREX1-usp45LS to generate pTREX1-sspA and pTREX1-sspB, resp., and the Ssp ***polypeptides*** were displayed on the cell surface of Lactococcus lactis MG1363. Lactococcal cells expressing similar levels of surface SspA or SspB ***polypeptide*** were then compared for their abilities to adhere to a range of antigen I/II ***polypeptide*** substrates. More than twice as many L. lactis cells expressing SspA bound to immobilized salivary agglutinin glycoprotein salivary agglutinin glycoprotein (SAG) as did L. lactis cells expressing SspB. In contrast, lactococci expressing SspB adhered twice as well as lactococci producing SspA to ***collagen*** type I and to Candida albicans. The binding of A. naeslundii to lactococci was only weakly enhanced by surface expression of Ssp ***polypeptides*** . L. lactis(pTREX1-sspB) cells bound in greater nos. to SAG than did Enterococcus faecalis JH2-2 cells expressing SspB from pAM401EB-5. The results suggest that SspA and SspB have markedly different binding affinities for their oral substrates and thus may function to promote site diversity in colonization by S. gordonii.

L14 ANSWER 30 OF 43 USPATFULL

AN 97:120738 USPATFULL
TI * *Polynucleotide*** encoding saliva binding * *protein***
IN Hodgson, John Edward, Malvern, PA, United States
Burnham, Martin Karl Russell, Norristown, PA, United States
PA SmithKline Beecham, p.l.c., Brentford, England (non-U.S. corporation)
PI US 5700928 19971223
AI US 1996-729202 19961015 (8)
PRAI GB 1995-21147 19951016
GB 1996-4599 19960304
GB 1996-16136 19960801
DT Utility
EXNAM Primary Examiner: Housel, James C.; Assistant Examiner: Bui, Phuong T.
LREP Gimmi, Edward R., King, William T., Lentz, Edward T.
CLMN Number of Claims: 36
ECL Exemplary Claim: 1
DRWN 4 Drawing Figure(s); 4 Drawing Page(s)
LN.CNT 1320
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Saliva binding ***protein*** ***polypeptides*** and ***DNA*** (RNA) of Staphylococcus aureus encoding such saliva binding ***protein*** and a procedure for producing such ***polypeptides*** by recombinant techniques is disclosed. Also disclosed are methods for utilizing such saliva binding ***protein*** for the treatment of infection, particularly bacterial infections. Antagonists against such saliva binding ***protein*** and their use as a therapeutic to treat infections, particularly bacterial infections are also disclosed. Also disclosed are diagnostics assays for detecting diseases related to the presence of saliva binding ***protein*** nucleic acid sequences and the ***polypeptides*** in a host. Also disclosed are diagnostic assays for detecting ***polynucleotides*** encoding saliva binding ***protein*** family and for detecting the ***polypeptide*** in a host.

L14 ANSWER 31 OF 43 USPATFULL
AN 97:66105 USPATFULL
TI Fibronectin binding ***protein***
IN Hook, Magnus, Birmigham, AL, United States
Jonsson, Klas, Studentvagen, Sweden
Lindberg, Kjell Martin, Kornvagen, Sweden
Signas, Christer, Hamnesplanaden, Sweden
PA Alfa-Laval Agri International Aktiebolag, Tumba, Sweden (non-U.S. corporation)
PI US 5652217 19970729
AI US 1994-340458 19941114 (8)
RLI Continuation of Ser. No. US 1992-974181, filed on 10 Nov 1992, now

abandoned which is a division of Ser. No. US 1990-520808, filed on 9 May 1990, now patented, Pat. No. US 5175096
PRAI SE 1989-1687 19890511
DT Utility
EXNAM Primary Examiner: Low, Christopher S. F.
LREP Burns, Doane, Swecker & Mathis
CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN 8 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 1101
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to a new recombinant hybrid- ***DNA*** -molecule comprising a nucleotide sequence from S. aureus coding for a ***protein*** , or ***polypeptide*** , having fibronectin binding properties.

L14 ANSWER 32 OF 43 USPATFULL
AN 97:38496 USPATFULL
TI Anti-fungal methods and materials
IN Little, II, Roger G., Benicia, CA, United States
Lim, Edward, Walnut Creek, CA, United States
Scannon, Patrick J., San Francisco, CA, United States
Lambert, Jr., Lewis J., Fremont, CA, United States
PA Xoma Corporation, Berkeley, CA, United States (U.S. corporation)
PI US 5627153 19970506
AI US 1995-372105 19950113 (8)
RLI Continuation-in-part of Ser. No. US 1994-273540, filed on 11 Jul 1994, now abandoned which is a continuation-in-part of Ser. No. US 1994-209762, filed on 11 Mar 1994 which is a continuation-in-part of Ser. No. US 1994-183222, filed on 14 Jan 1994, now abandoned
DT Utility
EXNAM Primary Examiner: Tsang, Cecilia J.; Assistant Examiner: Mohamed, Abdel A.
LREP Marshall, O'Toole, Gerstein, Murray & Borun
CLMN Number of Claims: 16
ECL Exemplary Claim: 1
DRWN 8 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 2065
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to methods for treating fungal infection comprising administering to a subject suffering from a fungal infection a bactericidal/permeability-inducing (BPI) ***protein*** product.

L14 ANSWER 33 OF 43 CAPLUS COPYRIGHT 2001 ACS
AN 1996:195920 CAPLUS

DN 124:310547
TI A two-domain mechanism for group A ***streptococcal** adherence through ***protein*** F to the extracellular matrix
AU Ozeri, Vered; Tovi, Aviva; Burstein, Israel; Natanson-Yaron, Shira; Caparon, Michael G.; Yamada, Kenneth M.; Akiyama, Steven K.; Vlodavsky, Israel; Hanski, Emanuel
CS Dep. Clinical Microbiology, Hebrew Univ.-Hadassah Med. Sch., Jerusalem, 91010, Israel
SO EMBO J. (1996), 15(5), 989-98
CODEN: EMJODG; ISSN: 0261-4189
DT Journal
LA English
AB ***Streptococcus*** pyogenes binds to the extracellular matrix (ECM) and a variety of host cells and tissues, causing diverse human diseases. ***Protein*** F, a S. pyogenes adhesin that binds fibronectin (Fn), contains 2 binding domains: a repeated domain (RD2) and an addnl. domain (UR) located immediately N-terminal to RD2. Both domains are required for maximal Fn binding. This study characterized RD2 and UR precisely and compared their functions and binding sites in Fn. The minimal functional unit of RD2 is of 44 amino acids, with contributions from 2 adjacent RD2 repeats flanked by a novel 'MGGQSES' motif. RD2 binds to the N-terminal fibrin binding domain of Fn. UR contains 49 amino acids, of which 6 are from the first repeat of RD2. It binds to Fn with higher affinity than RD2, and recognizes a larger fragment that contains fibrin and ***collagen*** ***binding*** domains. Expression of UR and RD2 independently on the surface-exposed region of unrelated ***streptococcal*** ***protein*** demonstrates that both mediate adherence of the bacteria to the ECM. A mechanism of adherence of a pathogen is described that involves 2 pairs of sites located on a single adhesin mol. and directed at the same host receptor.

L14 ANSWER 34 OF 43 USPATFULL
AN 95:71464 USPATFULL
TI Fibronectin binding peptide
IN Hook, Magnus, 129 Stevens Hill Cir., Birmingham, AL, United States 35244
McGavin, Martin, 1717 Beacon Crest Cir., Birmingham, AL, United States 35209
Raucci, Guiseppe, Via Tito Speri 10, I-00040 Pomezia, Rome, Italy
PI US 5440014 19950808
AI US 1994-234622 19940428 (8)
RLI Continuation of Ser. No. US 1993-55783, filed on 3 May 1993, now abandoned which is a continuation of Ser. No. US 1992-846995, filed on 8 Jun 1992, now abandoned
PRAI SE 1990-2617 19900810

DT Utility
EXNAM Primary Examiner: Warden, Jill; Assistant Examiner: Marshall, S. G.
LREP Burns, Doane, Swecker & Mathis
CLMN Number of Claims: 1
ECL Exemplary Claim: 1
DRWN 6 Drawing Figure(s); 6 Drawing Page(s)
LN.CNT 679
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A fibronectin binding peptide having the structure R'-PSYQFGGHNS VDFEEDT-R.sup.2 wherein R' is hydrogen, K or DK, and R.sup.2 is hydroxy, L, LP or LPK is disclosed. The fibronectin binding proteins of the present invention may be used, for example, for vaccination of ruminants against mastitis caused by Staphylococcal infections, for the treatment of wounds, e.g., for blocking ***protein*** receptors or for immunization (vaccination) against infection by bacterial strains, and for diagnosis of bacterial infections caused by Staphylococci strains.

L14 ANSWER 35 OF 43 USPATFULL
AN 95:43181 USPATFULL
TI Fibronectin binding ***protein*** -encoding ***DNA***
IN Hook, Magnus, Birmingham, AL, United States
Lindberg, Kjell M., Upsala, Sweden
Lindgren, Per-Eric, Upsala, Sweden
Signas, Lars C., Uppsala, Sweden
PA Alfa-Laval Agri International Aktiebolag, Tumba, Sweden (non-U.S. corporation)
PI US 5416021 19950516
AI US 1993-125222 19930923 (8)
RLI Continuation of Ser. No. US 1992-973551, filed on 9 Nov 1992, now abandoned which is a continuation of Ser. No. US 1989-352949, filed on 17 May 1989, now abandoned
PRAI SE 1988-1894 19880520
DT Utility
EXNAM Primary Examiner: Stone, Jacqueline; Assistant Examiner: Stanton, Brian R.
LREP Burns, Doane, Swecker & Mathis
CLMN Number of Claims: 8
ECL Exemplary Claim: 3
DRWN 10 Drawing Figure(s); 9 Drawing Page(s)
LN.CNT 441
AB The present invention relates to new recombinant ***DNA*** -molecules comprising nucleotide sequences of S. dysgalactiae encoding for at least one ***protein*** or ***polypeptide*** having fibronectin binding property.

L14 ANSWER 36 OF 43 CAPLUS COPYRIGHT 2001 ACS
AN 1995:393059 CAPLUS
DN 122:156198
TI Group B ***streptococci*** adhere to a variant of fibronectin attached to a solid phase
AU Tamura, Glen S.; Rubens, Craig E.
CS Children's Hosp. Med. Cent., Univ. Washington, Seattle, WA, 98105, USA
SO Mol. Microbiol. (1995), 15(3), 581-9
CODEN: MOMIEE; ISSN: 0950-382X
DT Journal
LA English
AB Group B ***streptococci*** (GBS) are the leading cause of neonatal pneumonia and meningitis. Adherence of GBS to host tissues may play and important role in the pathogenesis of infection. The host mols. which mediate GBS adherence to host tissues are unknown. Many bacterial pathogens adhere to fibronectin, an important component of the extracellular matrix (ECM). Some pathogens adhere to both immobilized and sol. fibronectin, while others adhere to immobilized fibronectin, but not to sol. fibronectin. Previous data indicated that GBS do not adhere to sol. fibronectin. We studied the ability of GBS to adhere to immobilized fibronectin. Forty-five per cent of the input inoculum of COH1, a virulent GBS isolate, adhered to fibronectin immobilized on polystyrene. COH1 did not adhere to the other ECM proteins tested (laminin, type 1 ***collagen*** , vitronectin, and tenascin). Nine out of nine GBS strains from human sources tested adhered specifically to fibronectin at levels varying from 4-60%. We considered the possibility that GBS were adherent to a contaminant in the fibronectin prepn. Properties of fibronectin, including the presence of an immunol. epitope of fibronectin and ***binding*** to ***collagen*** , were verified to be properties of the mol. to which GBS adhere. COH1 adhered to fibronectin captured by a monoclonal antibody to fibronectin (FN-15), confirming that the mol. to which GBS adhere bears immunol. determinants of fibronectin. Adherence of COH1 to fibronectin was inhibited by ***collagen*** , confirming that the mol. to which GBS adhere binds to ***collagen*** . These data strongly suggest that GBS adhere to fibronectin, and not to a contaminant. ***Protein*** blot anal. revealed that GBS were adherent to a high-mol.-wt. variant of non-reduced fibronectin monomers and dimers. GBS did not adhere to reduced fibronectin monomers. We conclude that GBS adhere to a variant of plasma fibronectin when attached to a solid phase.

L14 ANSWER 37 OF 43 CAPLUS COPYRIGHT 2001 ACS
AN 1995:311506 CAPLUS
DN 122:75071
TI Isolation and characterization of a novel ***collagen*** - ***binding*** ***protein*** from ***Streptococcus*** pyogenes

strain 6414
AU Visai, Livia; Bozzini, Silvia; Raucci, Giuseppe; Toniolo, Antonio; Speziale, Pietro
CS Dep. Biochem., Univ. Pavia, Pavia, I-227100, Italy
SO J. Biol. Chem. (1995), 270(1), 347-53
CODEN: JBCHA3; ISSN: 0021-9258
DT Journal
LA English
AB In this report the authors have analyzed the ***binding*** of ***collagen*** to ***Streptococcus*** pyogenes strain 6414. This binding was rapid, specific, and involved a limited no. of receptor mols. (11,600 copies per cell). When the proteins in a ***streptococcal*** lysate were blotted onto a nitrocellulose filter and probed with 125I-labeled ***collagen*** , a prominent ***collagen*** - ***binding*** ***protein*** of 57 kDa was identified as well as minor 130-150-kDa components. The major 57-kDa ***protein*** was isolated by affinity chromatog. on ***collagen*** -Sepharose followed by gel filtration chromatog. The 57-kDa ***protein*** purified from S. pyogenes was used to raise a monospecific antibody which also reacted with a ***collagen*** - ***binding*** ***protein*** of similar mol. size isolated from ***Streptococcus*** zooepidemicus. The two ***collagen*** - ***binding*** proteins from ***streptococci*** have a similar ***amino*** ***acid*** compn. and isoelec. points. Isolated ***collagen*** - ***binding*** ***protein*** was specifically recognized by 125I- ***collagen*** in a solid-phase ***binding*** assay and displayed an affinity for the ligand quite similar to that exhibited by intact bacteria (Kd = 3.1 vs. 3.5.times.10-9 M, resp.). Surface-labeled bacteria attached to microtiter wells coated with different ***collagen*** types and the 57-kDa ***protein*** blocked the ***adhesion*** to ***collagen*** substrate. The authors propose that the 57-kDa ***protein*** is an adhesin involved in the attachment of ***streptococci*** to host tissues.

L14 ANSWER 38 OF 43 CAPLUS COPYRIGHT 2001 ACS
AN 1994:51458 CAPLUS
DN 120:51458
TI ***Collagen*** mediates ***adhesion*** of ***Streptococcus*** mutans to human dentin
AU Switalski, Lech M.; Butcher, Wade G.; Caufield, Page C.; Lantz, Marilyn S.
CS Sch. Dent. Med., Univ. Pittsburgh, Pittsburgh, PA, 15261, USA
SO Infect. Immun. (1993), 61(10), 4119-25
CODEN: INFIBR; ISSN: 0019-9567
DT Journal
LA English
AB Some strains of ***Streptococcus*** mutans were found to recognize and

bind ***collagen*** type I. ***Binding*** of 125I-labeled ***collagen*** type I was specific in heat ** collagen* * types I and II, but not unrelated proteins, were able to inhibit binding to the labeled ligand to bacteria. ***Collagen*** ***binding*** to S. mutans was partially reversible and involved a limited no. of bacterial binding sites per cell. S. mutans UA 140 cells bound ***collagen*** type I with high affinity. The no. of binding sites per cell was 4 .times. 104. ***Collagen*** - ***binding*** strains of S. mutans adhered to ***collagen*** -coated surfaces as well as to pulverized root tissue. S. mutans strains that did not bind the sol. ligand were unable to adhere to these substrata. Adherence to ***collagen*** -coated surfaces could be inhibited with ***collagen*** or clostridial collagenase-derived ***collagen*** peptides. S. mutants UA 140 bound significantly less 125I- ***collagen*** type I following treatment with peptidoglycan-degrading enzymes. These enzymes released a ***collagen*** - ***binding*** ***protein*** (***collagen*** receptor) with a relative mol. size of 16 kDa. Apparently, ***collagen*** mediates ***adhesion*** of S. mutans to dentin. This interaction may target ***collagen*** - ***binding*** strains of S. mutans to dentin in the oral cavity and may play a role in the pathogenesis of root surface caries.

L14 ANSWER 39 OF 43 USPATFULL
AN 92:106759 USPATFULL
TI ***DNA*** encoding a fibronectin binding ***protein*** as well as its preparation
IN Hook, Magnus, Birmingham, AL, United States
Jonsson, Klas, Studentvagen, Sweden
Lindberg, Kjell M., Kornvagen, Sweden
Signas, Christer, Hamnesplanaden, Sweden
PA Alfa-Laval Agri International Aktiebolag, Tumba, Sweden (non-U.S. corporation)
PI US 5175096 19921229
AI US 1990-520808 19900509 (7)
PRAI SE 1989-1687 19890511
DT Utility
EXNAM Primary Examiner: Lacey, David L.; Assistant Examiner: Ulm, John D.
LREP Burns, Doane, Swecker & Mathis
CLMN Number of Claims: 6
ECL Exemplary Claim: 1
DRWN 5 Drawing Figure(s); 8 Drawing Page(s)
LN.CNT 1147
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to a new recombinant hybrid- ***DNA*** -molecule comprising a nucleotide sequence from S. aureus coding for a

protein , or ***polypeptide*** , having fibronectin binding properties.

L14 ANSWER 40 OF 43 USPATFULL
AN 92:42541 USPATFULL
TI Method for treating benign prostatic hypertrophy
IN Gokcen, Muharrem, Minneapolis, MN, United States
Guy, Terry J., Chaska, MN, United States
PA Immunolytics, Inc., Minneapolis, MN, United States (U.S. corporation)
PI US 5116615 19920526
AI US 1991-707628 19910530 (7)
RLI Continuation of Ser. No. US 1989-429966, filed on 31 Oct 1989, now abandoned which is a continuation-in-part of Ser. No. US 1989-303809, filed on 27 Jan 1989, now abandoned
DT Utility
EXNAM Primary Examiner: Stone, Jacqueline
LREP Merchant, Gould, Smith, Edell, Welter & Schmidt
CLMN Number of Claims: 19
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 3209
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The invention provides a composition and method for treating benign prostatic hypertropy in mammals so as to cause the dissolution and regression of hypertrophied prostatic tissue and thereby provide relief from the obstructive symptoms associated with the disease. The present composition preferably comprises a sterile pyrogen-free solution of the hydrolytic enzymes collagenase and hyaluronidase, a nonionic surfactant, and an antibiotic; all provided, in a pharmaceutically acceptable, buffered, isotonic, aqueous carrier. The present method preferably comprises the direct intraprostatic injection of a safe and therapeutically effective dose of the composition via the transurethral route of administration.

L14 ANSWER 41 OF 43 USPATFULL
AN 91:60799 USPATFULL
TI Methods for treating damaged corneal, uterine, or cartilage tissue
IN Kludas, Martin, Herthastrasse 22, D-1000 West Berlin-33, Germany, Federal Republic of
PI US 5036056 19910730
AI US 1990-500330 19900327 (7)
RLI Continuation of Ser. No. US 1987-70991, filed on 8 Jul 1987, now abandoned
DT Utility
EXNAM Primary Examiner: Kilby Scalzo, Catherine S.

LREP Pennie & Edmonds
CLMN Number of Claims: 31
ECL Exemplary Claim: 1
DRWN No Drawings
LN.CNT 850
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB Methods for treating damaged corneal, uterine or cartilage tissue comprising applying a therapeutically effective amount of a composition comprising:

(a) a sterile extracellular connective tissue matrix composition comprising collagens, proteoglycans, glycosaminoglycans and glycoproteins wherein said collagens, said proteoglycans, said glycosamino glycans, and said glycoproteins have each been extracted from an extracellular connective tissue matrix and are in their native structural form and

(b) a pharmaceutically acceptable carrier.

L14 ANSWER 42 OF 43 CAPLUS COPYRIGHT 2001 ACS
AN 1990:627897 CAPLUS
DN 113:227897
TI ***Streptococcus*** cricetus and ***Streptococcus*** rattus bind to different segments of ***collagen*** molecules
AU Liu, Tianjia; Gibbons, R. J.; Hay, D. I.
CS Dep. Microbiol., Forsyth Dent. Cent., Boston, MA, 02115, USA
SO Oral Microbiol. Immunol. (1990), 5(3), 143-8
CODEN: OMIMEE; ISSN: 0902-0055
DT Journal
LA English
AB Strains of S. cricetus and S. rattus exhibited striking differences in their ability to bind to different types of ***collagen*** . For example, S. cricetus AHT bound in highest nos. to hydroxyapatite (HA) treated with human type V ***collagen*** , while rat type I ***collagen*** was ineffective. In contrast, human type V ***collagen*** was least effective in promoting attachment of S. rattus LB-1, while treatment with rat of human type I ***collagen*** was effective. Adsorption of both species to human type I ***collagen*** -treated HA showed a high correlation with a Langmuir model. Ests. of adsorption parameters indicated there were greater nos. of binding sites with higher affinity for S. rattus LB-1 than for S. cricetus AHT. Treatment of HA with either the alpha 1 (1) or alpha 2 (1) ***polypeptide*** chains of ***collagen*** was effective in promoting ***adhesion*** of S. rattus LB-1 cells. In contrast, the alpha 2 (1) chain was more effective than the alpha 1 (1), chain for S.

cricetus AHT. These observations indicate that S. cricetus AHT and S. rattus LB-1 cells bind to different segments of ***collagen*** mols. ***Adhesion*** of both species was also promoted by ***collagen*** -rich fractions of human dentin.

L14 ANSWER 43 OF 43 CAPLUS COPYRIGHT 2001 ACS
AN 1985:484973 CAPLUS
DN 103:84973
TI Extracellular matrix proteins (fibronectin, laminin, and type IV ***collagen***) bind and aggregate bacteria
AU Vercellotti, G. M.; McCarthy, J. B.; Lindholm, P.; Peterson, P. K.; Jacob, H. S.; Furcht, L. T.
CS Dep. Med., Univ. Minnesota, Minneapolis, MN, 55455, USA
SO Am. J. Pathol. (1985), 120(1), 13-21
CODEN: AJPAA4; ISSN: 0002-9440
DT Journal
LA English
AB The normal microbial colonization of sites in body tissues by certain bacteria requires that the bacteria first bind to extracellular secreted constituents, cell-surface membranes, or cell matrixes. Two interactions of a variety of bacteria with the cell matrix noncollagenous proteins fibronectin and laminin and with basement membrane (type IV) ***collagen*** , were studied. Adherence of bacteria to matrix proteins coated on tissue culture wells was examd. with the use of radiolabeled bacteria. Staphylococcus aureus, ***Streptococcus*** pyrogenes, And S. sanguis bound well to fibronectin, laminin, and type IV ***collagen*** , whereas a variety of gram-neg. organisms did not bind. The interaction of sol. laminin, fibronectin, and type IV ***collagen*** with bacteria was monitored by nephelometry with the use of a platelet aggregometer. S. aureus Aggregated in response to fibronectin, laminin, or type IV ***collagen*** . In contrast, gram-neg. organisms did not aggregate with these proteins. Fibronectin, laminin, and type IV ***collagen*** can bind and aggregate certain gram-pos. bacteria, and this binding is dependent on the surface characteristics of the organism. The ***adhesion*** mols. may play a role in the normal colonization of sites by microorganisms and in invasion during infections.

=> d 13 17 29 clm

NO VALID FORMATS ENTERED FOR FILE 'CAPLUS'
In a multifile environment, each file must have at least one valid format requested. Refer to file specific help messages or the STNGUIDE file for information on formats available in individual files.

REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):d clm 13 17

'D' IS NOT A VALID FORMAT
'13' IS NOT A VALID FORMAT
'17' IS NOT A VALID FORMAT
In a multifile environment, a format can only be used if it is valid
in at least one of the files. Refer to file specific help messages
or the STNGUIDE file for information on formats available in
individual files.
REENTER DISPLAY FORMAT FOR ALL FILES (FILEDEFAULT):.

L14 ANSWER 13 OF 43 USPATFULL
AN 2000:4645 USPATFULL
TI Cell surface ***protein*** compounds
IN Hodgson, John Edward, Malvern, PA, United States
Burnham, Martin Karl Russell, Norristown, PA, United States
PA SmithKline Beecham plc, United Kingdom (non-U.S. corporation)
PI US 6013482 20000111
AI US 1996-730261 19961015 (8)
DT Utility
LN.CNT 1255
INCL INCLM: 435/069.300
INCLS: 435/252.300; 435/320.100; 435/325.000; 536/023.700
NCL NCLM: 435/069.300
NCLS: 435/252.300; 435/320.100; 435/325.000; 536/023.700
IC [6]
ICM: C12P021-06
ICS: C12N001-20; C12N015-00; C07H021-04
EXF 536/23.7; 435/320.1; 435/69.3; 435/252.3; 435/325
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L14 ANSWER 17 OF 43 USPATFULL
AN 1999:65197 USPATFULL
TI ***DNA*** encoding fibronectin and fibrinogen binding
protein from group A ***streptococci***
IN Rocha, Claudia, New York, NY, United States
Fischetti, Vincent A., West Hempstead, NY, United States
PA The Rockefeller University, New York, NY, United States (U.S.
corporation)
PI US 5910441 19990608
AI US 1996-714402 19960916 (8)
DT Utility
LN.CNT 959
INCL INCLM: 435/320.100

INCLS: 435/325.000; 536/023.700
NCL NCLM: 435/320.100
NCLS: 435/325.000; 536/023.700
IC [6]
ICM: C12N015-00
ICS: C12N005-00; C07H021-04
EXF 536/23.7; 435/320.1; 435/325
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L14 ANSWER 29 OF 43 CAPLUS COPYRIGHT 2001 ACS
AN 1998:649589 CAPLUS
DN 129:341523
TI Binding properties of ***Streptococcus*** gordonii SspA and SspB (antigen I/II family) ***polypeptides*** expressed on the cell surface of Lactococcus lactis MG1363
AU Holmes, Ann R.; Gilbert, Christophe; Wells, Jeremy M.; Jenkinson, Howard F.
CS Department of Oral Sciences and Orthodontics, University of Otago, Dunedin, N. Z.
SO Infect. Immun. (1998), 66(10), 4633-4639
CODEN: INFIBR; ISSN: 0019-9567
PB American Society for Microbiology
DT Journal
LA English

=> d clm 13 17

L14 ANSWER 13 OF 43 USPATFULL
CLM What is claimed is:
1. An isolated ***polynucleotide*** segment encoding SEQ ID NO: 1.

2. An isolated nucleic acid segment comprising a nucleotide sequence which is fully complementary to the ***polynucleotide*** of claim 1.

3. An isolated vector comprising the ***polynucleotide*** segment of claim 1.

4. An isolated vector comprising the nucleic acid segment of claim 2.

5. An isolated host cell comprising the vector of claim 3.

6. An isolated host cell comprising the vector of claim 4.

7. A process for producing a ***polypeptide*** encoded by said ***polynucleotide*** segment comprising culturing the host cell of claim 5 under conditions sufficient for the production of said ***polypeptide*** .

8. An isolated ***polynucleotide*** segment encoding a mature ***polypeptide*** expressed by a ***polynucleotide*** comprising SEQ ID NO: 2 in deposited strain NCIMB 40771.

9. An isolated nucleic acid segment comprising a nucleotide sequence which is fully complementary to the ***polynucleotide*** segment of claim 8.

10. An isolated vector comprising the ***polynucleotide*** segment of claim 8.

11. An isolated vector comprising the nucleic acid segment of claim 9.

12. An isolated host cell comprising the vector of claim 10.

13. An isolated host cell comprising the vector of claim 11.

14. A process for producing the mature ***polypeptide*** comprising culturing the host cell of claim 12 under conditions sufficient for the production of said ***polypeptide*** .

L14 ANSWER 17 OF 43 USPATFULL
CLM What is claimed is:

1. A purified ***DNA*** fragment encoding the ***Streptococcal*** fibrinogen and fibronectin binding ***protein*** (SFFBP-12)(SEQ ID NO: 2).

2. A ***DNA*** according to claim 1, wherein the ***DNA*** comprises the sequence of the sffbp-12 gene (SEQ ID NO: 1).

3. A replicable expression vector comprising the ***DNA*** of claim 1.

4. An isolated host cell transformed with the vector of claim 3.

5. A ***DNA*** according to claim 1, operably linked to one or more elements selected from the group consisting of a promoter, a transcription enhancer element, a termination signal, a translation signal, and a combination of two or more of these elements.

6. A ***DNA*** according to claim 5, further comprising a selectable marker.

7. A ***DNA*** according to claim 1, wherein the ***DNA*** consists of the sequence of the sffbp-12 gene (SEQ ID NO: 1).

8. A replicable expression vector comprising the ***DNA*** of claim 7.

9. An isolated host cell transformed with a vector according to claim 8.

10. A ***DNA*** according to claim 7, further comprising one or more elements selected from the group consisting of a promoter, a transcription enhancer element, a termination signal, a translation signal, and a combina

---Logging off of STN---

tion of two or more of these elements.

11. A ***DNA*** according to claim 10, further comprising a selectable marker.

=>

Executing the logoff script...

=> LOG Y

=>

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	182.01	182.16

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-16.46	-16.46

STN INTERNATIONAL LOGOFF AT 15:59:13 ON 21 JUN 2001